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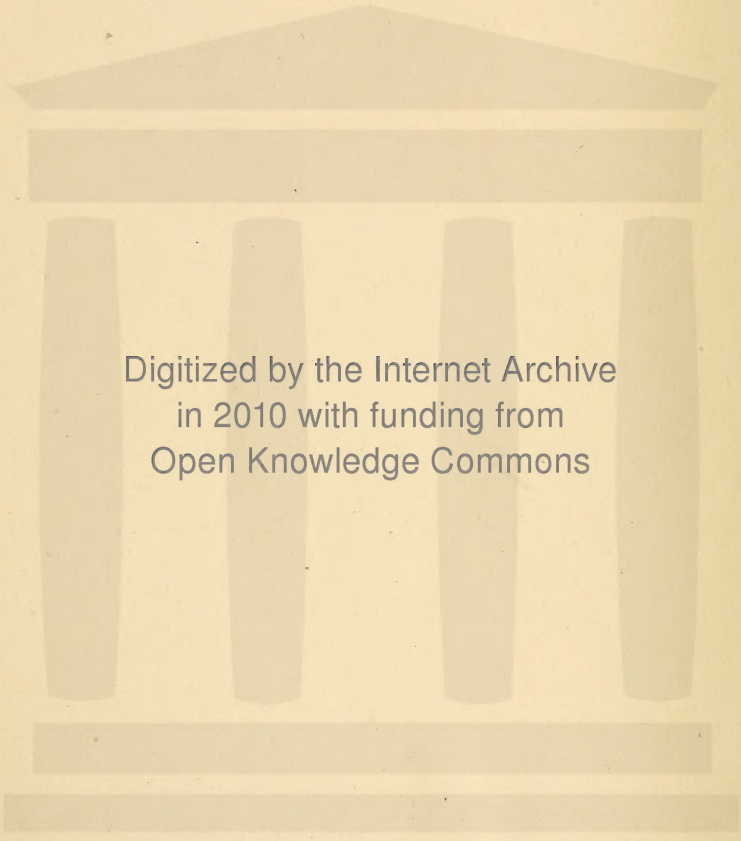
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TWENTIETH CENTURY PRACTICE

AN INTERNATIONAL ENCYCLOPEDIA

OF

MODERN MEDICAL SCIENCE

BY

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EDITED BY

THOMAS L. STEDMAN, M.D.
NEW YORK CITY

IN TWENTY VOLUMES

VOLUME VI.

DISEASES OF THE RESPIRATORY ORGANS.

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CONTRIBUTORS TO VOLUME VI.

WINSLOW ANDERSON, M.D., M.R.C.P., M.R.C.S., San Francisco.

Vice-President of the California State Board of Health; formerly Adjunct to the Chair of Principles and Practice of Medicine, Medical Department of the University of California.

FRANCKE H. BOSWORTH, M.D., New York.

Professor of Diseases of the Throat, Bellevue Hospital Medical College; Physician-in-Charge of the Clinic for Diseases of the Throat in the Outdoor Department of Bellevue Hospital.

ALBERT H. BUCK, M.D., New York.

Consulting Aural Surgeon to the New York Eye and Ear Infirmary and to the Presbyterian Hospital; Professor of Diseases of the Ear, Medical Department of Columbia College.

GEORGE A. GIBSON, M.D., D.Sc., F.R.C.P. Ed., Edinburgh.

Assistant Physician, Royal Infirmary; Lecturer on the Principles and Practice of Medicine, Minto House School.

PROSSER JAMES, M.D., M.R.C.P., M.R.C.S., London.

Formerly Lecturer on Materia Medica and Therapeutics, London Hospital; Physician to the Hospital for Diseases of the Throat and to the North London Consumptive Hospital, and Consulting Physician to the Children's Home Infirmary, Victoria Park.

E. J. MOURE, M.D., Bordeaux.

Professor of Laryngology, Otology, and Rhinology, University of Bordeaux; Editor of the Revue de Laryngologie, d'Otologie et de Rhinologie.

SIR THOMAS GRAINGER STEWART, M.D., F.R.S., F.R.C.P. Ed., Edinburgh.

Professor of Medicine and of Clinical Medicine, University of Edinburgh; Physician to Her Majesty the Queen in Scotland.

JONATHAN WRIGHT, M.D., Brooklyn.

Clinical Professor of Diseases of the Nose and Throat in the Woman's Medical College of the New York Infirmary.

DISEASES OF THE NOSE.

BY

PROSSER JAMES,

LONDON.

DISEASES OF THE NOSE.

It is unnecessary in a work of this kind to describe the bony and cartilaginous framework of the nose. All text-books of anatomy contain full details. The conformation of this important feature is such that morbid processes affecting it are either quite open to view, or so hidden as to require special apparatus to bring them within the range of vision. The former diseases may be classed as external. The latter, or internal, affecting the nasal passages and requiring special appliances both for diagnosis and treatment, are those with which we are chiefly concerned in this article, and the external diseases of the nose will be passed over briefly. They comprise affections of the skin and subcutaneous tissues, most of which are committed to the dermatologist, and certain deformities and injuries which fall to the care of the surgeon.

EXTERNAL DISEASES.

Acne.

Acne in various forms implicates the nose, but what is called acne rosacea essentially belongs to it. It is often due to alcoholic indulgence and has obtained the name of "tippler's nose," but it may occur from other causes. In this disease the integument becomes highly injected and the venules put on a varicose appearance. There is redness with swelling, chiefly of the tip, causing considerable disfigurement. There is a true hypertrophy of the skin, the sebaceous follicles are usually enlarged, and a shiny or greasy state of the surface is produced. Later on crusts and scales form, and a blotchy or tubercular appearance is observed.

Treatment.—Slight cases may be benefited by strong lead lotion or weak solution of corrosive sublimate. A collodion containing ichthyol and resorcin may be painted on daily for three days after any pustules that are present have been punctured. In five or six days the collodion cracks and separates, when the application may be repeated or iodoform collodion may be substituted. A sulphur ointment may follow. Indeed sulphur maintains its repute in this as in acne

vulgaris. In severe cases subcutaneous division of the enlarged vessels has sometimes been followed by diminution, but multiple puncture or electrolysis will be preferable. It is useless to resort to such measures without a reform in the diet and regimen of most patients.

Furuncle.

When a boil attacks the nose it causes great pain and disfigurement. It begins in the subcutaneous cellular tissue or sometimes beneath the perichondrium but quickly involves the skin and fibrocartilage, giving rise to the swelling and throbbing and tension characteristic of inflammation in unyielding structures.

It most commonly affects young or middle-aged persons of intemperate habits, but is also met with in the feeble and anæmic. When it attacks the old they will mostly be found to be free livers and troubled with constipation.

Treatment.—In the early stage lead lotion has long been used, but a disinfecting compress kept constantly on the part is perhaps better. Chloral hydrate, one-half ounce dissolved in one ounce each of glycerin and water, makes a good lotion of the kind and relieves the pain. Boric or salicylic lotions may be preferred by some. Carbolic acid as a compress or injected into the substance of boils is a favorite treatment and may be adopted in this situation. Such injection should be made from the nostril. So too, if an opening become necessary for the evacuation of pus the puncture should be made in the nostril in order to avoid the formation of an external scar.

Verruca.

A simple wart on the nose is a disfigurement which some find intolerable. There is no reason for treating it differently when in this locality than when on other parts of the surface, except that the more severe caustics should not be used. If acetic or carbolic acid be applied to the surface every night, a cure may be effected with scarcely a mark remaining. The surrounding skin should be protected with vaseline, then the acid dissolves the epidermis and reaches the vascular papillæ.

Hypertrophy.

When this affects the skin and subcutaneous tissue, producing an elephantoid enlargement of the tip, it is often termed lipoma, but erroneously so, for the neoplasm is really poor in fat and composed of dense fibrous tissue, containing few blood-vessels. Near the surface, and sometimes projecting from it are enlarged sebaceous glands dis-

tended with thickened secretion. Altogether the growth resembles elephantiasis elsewhere. It is a painless enlargement, which often occurs in acne rosacea of severe type and long standing. Sometimes it is lobulated or pedunculated and may grow to the size of a hen's egg. It does not involve the cartilages.

The only available *treatment* is careful dissection of the tumor from the perichondrium. Sometimes all the tissues of the tip of the nose hypertrophy, and for this some surgeons have excised wedge-shaped portions so as to admit of a proper adaptation of the cut surfaces.

Sebaceous Tumors.

Sebaceous tumors on the tip or alæ are also met with occasionally. The only treatment for them is excision.

Nævus.

Nævus may implicate the nose. Vaccination has removed small nævi but the mark remains. Ligature leaves a worse scar. Sodium ethylate suffices to remove some. Injections are not devoid of danger. The best method of dealing with them is by electrolysis or the galvano-cautery.

Rodent Ulcer.

Rodent ulcer so often commences on the nose as to require mention. After a long period of quiescence the pimple ulcerates. Slowly the ulcer spreads, involving every tissue in its destructive march until frightful ravages are produced. The nose may be destroyed, as may also the eyeball, much of the face, and even the upper jaw. When the forehead is involved the dura mater may be exposed. The striking feature of the disease is that although fresh growth is going on, ulceration keeps pace with the deposit, so that there is no swelling but a continuous diminution of the size of the parts invaded. The surface of the ulcer is uneven, covered with imperfect granulations, and presents a glistening appearance of a mottled yellowish-red color. The margin is deeper red or purplish, rather hard, and usually undermined. A thin, purulent discharge often mixed with blood is present. The disease has been classed by some with cancer and the microscopic appearances resemble in some points those of epithelioma, but in other points they differ. Then rodent ulcer may continue its slow course for years, does not become generalized by an infective process, but is rather a local disease which, if entirely removed, may not return.

Treatment, therefore, is removal or destruction, and this must be thorough. Where possible the knife is the effective remedy, and the earlier it is employed the better. Any part that cannot be removed by the knife should be destroyed by zinc chloride.

Lupus.

The nose is a favorite point of attack of the several forms of disease to which the name lupus has been given.

Lupus erythematosus is characterized by chronic congestion accompanied by small-celled new growth and followed by interstitial absorption, leaving a scar. The new growth develops immediately under the epidermis, round the orifices of the ducts and follicles, destroying the normal tissue, which is partly removed by absorption and partly by fatty degeneration. The invasion is usually in the form of small red patches, which may remain for a long time unaltered or only deepening in color but eventually become covered with dirty-looking, thin crusts, closely adherent by means of processes extending into the dilated orifices of the sebaceous follicles, as can be seen on removal. Later on white scars succeed the purple-red color. Confluence between the patches occurs and results in curious shapes, such as the "bat's-wing" form produced by the union of two patches across the nose.

Lupus vulgaris is also a very chronic disease characterized by infiltration of small round cells into the skin or mucous membrane, for it attacks both. There is interstitial absorption or ulceration. A small patch or two—perhaps no larger than a pin's head, perhaps several times larger—level with the skin or somewhat raised, may for a long time remain as the only symptom. Somewhat larger blotches are occasionally the first observed. Other patches form, and eventually confluence between some of them takes place, so that a considerable surface may be invaded from which thin epidermic scales are pretty constantly shed—*lupus exfoliativus*. At a later stage interstitial absorption sets in, and the elevated patch or tubercle is succeeded by a bluish-white scar, or else the tubercles soften and a clean-cut, pink-edged ulcer with a granulating base results. The purulent secretion hardens and a greenish-yellow scale is produced, but beneath this the ulceration spreads and deepens, attacking any tissues it approaches. This is *lupus exedens*, which, though it may attack any part of the surface, so constantly begins on the nose as to make that location characteristic. The tip or one of the alæ is most often the first part to be invaded, though sometimes the nostril is attacked from the inside. The ulceration often destroys the nose

and large parts of the lips and cheeks, producing the most frightful appearance, and might well give rise to the term *noli me tangere*.

Etiology.—Lupus attacks women more frequently than men and country people more often than town-dwellers. It is not hereditary. Lupus erythematosus is rare before puberty. No age of adult life is exempt, but usually it begins between the twentieth and thirtieth year. On the other hand lupus vulgaris generally begins early, and often by the second or third year, and from that age up to puberty when the liability declines rapidly. It very rarely begins after the thirtieth year.

Pathology.—Infiltration by vast numbers of round cells gradually destroys the tissue in which the deposit occurs. The new growth is highly vascular and tends to follow the distribution of the blood-vessels. Virchow compares it to granulation tissue. It insinuates itself between the bands of connective tissue and the glands. It is deposited in distinct nodules, resembling gray tubercle (Friedländer) in which giant cells are found, so confirming the view of those who consider it allied to tuberculosis. Lupus is certainly met with associated with phthisis but more commonly with enlarged or suppurating cervical glands and other so-called scrofulous symptoms. Micrococci have been described by some observers, but the tubercle bacillus would appear to be most probably the cause. The deeper cells are usally in a state of fatty degeneration and contain a reddish pigment. Indeed, the lupus tissue may all become fatty and then be absorbed without breach of the epidermis which is merely thickened, but more commonly the cuticle gives way and ulceration is thus established. *Pari passu* with the loss of tissue caused by ulceration, the proliferation of round cells is more active so that there appears to be a rapid new growth. Cicatrization follows and the contraction gives rise to great deformity. In a few cases epithelioma has appeared on an old-standing lupus or in the cicatrix.

Diagnosis.—Lupus erythematosus is distinguished from lupus vulgaris by the age of the patient, the superficial character of the lesion, its sebaceous crusts, and later its whitish superficial scars. Lupus exedens is at some period exceedingly likely to be mistaken for syphilis, especially when it begins rather late or commences within the nostril, where even Billroth regarded error as unavoidable. The slowness of progress, the history, and resistance to specific treatment may assist, as may also examination of other parts of the body for marks of syphilis. The microscope must be appealed to in the differentiation between carcinoma, sarcoma, tubercle, and lupus.

Prognosis.—Lupus must always be regarded as a grave disease, not as regards life, but in respect to the deformity it produces and the

tendency to relapse which it displays. When only mucous membrane is involved, a cure may be confidently expected and in this tissue a scar is scarcely ever left. Any indurated hypervascular tissue that may remain must be regarded as a likely locality for a relapse. Any tendency to extend in the direction of the pharynx is unfavorable. On the skin the extent of ulceration and the cicatrix add to the gravity.

Treatment.—Removal or destruction of the lupus tissue is the only method. The first may be accomplished mechanically by Volkmann's spoons freely used under local anæsthesia. But even after thorough scraping caustics should be employed to follow up the prolongations of lupus tissue along the hair follicles and other vascular structures. If caustics alone be trusted to, the most penetrating should be selected. Zinc chloride is one of the most reliable. More recently chromic and lactic acids have somewhat superseded it. Terchloracetic acid is equally available, and so are caustic potassa and silver nitrate. The above-mentioned liquids or solutions of the solids may be applied on tiny sponges or absorbent cotton. The pain caused is not of long duration. Lunar caustic used to be employed in the solid form, the softness of the new growth permitting the stick to be bored through it down to healthy tissue which resists a reasonable degree of force. The actual cautery and the galvano-cautery so disfigure the parts as to make it difficult to distinguish the point where healthy tissue is reached. Multiple scarification is employed in non-ulcerated lupus when the parts are swollen with diffuse cell infiltration. Although such tissue could be broken down by the spoon, multiple scarification or puncture is preferable as it suffices to destroy new vessels, induces absorption of the neoplasm, and causes less scarring than does scraping. In lupus erythematosus and even lupus exedens, when there is no ulceration and the disease is not advanced and quite superficial, mercurial plaster, iodine paint, pyrogallic-acid ointment (Hebra), and other mild measures may be useful. Sometimes soothing remedies are called for, and they should generally be employed between the applications of caustic or the scrapings. Constitutional treatment may be indicated by the condition of the patient, *e.g.*, cod-liver oil and tonics. Arsenic has been supposed to exercise a specially beneficial influence on the disease.

Rhinoscleroma.

This rare disease was first described by Hebra in 1870. Cases have since been recorded by Mikulicz, Geber, Weinlechner, Tantuzzi, Pellizzari, Neumann, Cornil and Alvarez, and others. Kaposi has treated of it at considerable length, and in October, 1895, Professor

Péan brought a most interesting case before the Academy of Medicine of Paris, where the subject was duly discussed. The disease in some features resembles lupus and is characterized by great thickening and induration. It usually begins at or close to the edge of the nostril and extends to the upper lip as well as to the nasal septum and passages. It appears as raised, smooth patches, so hard that they have been compared to ivory, but the color is natural or grayish pink, and they are tender on pressure though not otherwise painful. The patches enlarge and some may coalesce. They spread very slowly by infiltration and rarely ulcerate, though they continue to extend for years. The surrounding parts swell and become invaded, the march being more often along the mucous membrane than the skin, so that the disease in the course of years may pass through the nose and implicate the pharynx and larynx, or more rarely travel over the lip to the mouth.

Pathology.—The bacillus which seems to be the cause has been described by Chiari and in more detail by Cornil and Alvarez. The rods are thick and short, seldom above 3 μ in length but have been seen as long as 7 μ. They are enclosed, one or several, in a capsule and are difficult to stain. They can best be treated with methyl-violet B (Alvarez) in solution, with or without aniline, being left in the fluid for twenty-four hours. The bacilli are often seen lying in the large hyaline cells within the fibrous meshes of the tumor, but are also found free in the lymph vessels or in the interstices of the tissue. The growth on gelatin resembles Friedländer's pneumococcus, and some bacteriologists are still doubtful whether it is a distinct species, though when stained with basic aniline dye it is not decolorized by Gram's method, so far differing from Friedländer's bacillus, which is. Inoculation with cultures has given rise to the disease in animals (Stepanow, *Monatsheft für Ohrenheilkunde*, 1889).

Diagnosis.—From malignant disease it is easily distinguished by its dense, stony hardness and slow progress; from syphilis by the same characters and resistance to treatment.

Prognosis.—This is favorable as regards life unless the disease invade the larynx and demand tracheotomy, when it becomes serious.

Treatment, whether general or local, is without effect. Excision and caustics are both unavailing, as recurrence has always occurred. All that can be done is to meet complications. If the nasal passage becomes obstructed the growth may be removed or destroyed by caustic, and dilatation by laminaria bougies may be employed. None of these measures seems to stimulate to fresh growth. If the larynx should be invaded, tracheotomy must be done, as in a case reported by Schulthess. A case of cure by the constant application of a one-

per-cent. ointment of corrosive sublimate has been recorded by Doutrelepont. The rapid growth in this case, however, and some other points, as well as the fact of recovery, forcibly suggest that the case may have been syphilis.

Epithelioma.

Epithelioma of the nose is met with occasionally, commencing usually at the junction of the skin and mucous membrane as a warty patch, which may remain stationary for a long time but eventually passes on to the ulcerative stage and, unless excised, to the destruction of the organ. The glands generally are affected. The diagnosis from syphilis is most important, the history, effect of treatment, and multiple lesions of the latter being of great assistance. The progress and treatment do not differ from those of the same disease elsewhere.

Other malignant diseases generally commence from within and will be considered among the internal nasal affections.

Injuries.

Injuries of the nose may affect both the exterior and interior, and embrace all forms of wounds, fractures of the bones and dislocations, though these last are very rare. Injuries belong to the domain of surgery, as do the various plastic and other operations performed for the removal of deformities or the restoration of lost parts. They need not, therefore, be further considered except to point out the extreme importance, in recent cases, of the means for preventing disfigurement, whether by the exact coaptation of wounds, the introduction of plugs to prevent falling in of the nostril, or other contrivances to preserve as far as possible the natural appearance.

INTERNAL DISEASES.

We pass on now to the interior affections, and as these require the aid of the rhinoscope it will be convenient to describe here the use of that instrument.

Rhinoscopy.

Under this term we include any method of inspecting the nasal passages whether from before or from behind. In the former, or *anterior* rhinoscopy we have only to throw a beam of light into the dilated nostril in order to obtain a direct view of the interior. In *posterior* rhinoscopy, the procedure is more complicated, as we have to hold in

the fauces a mirror on which the light is focussed and in which we see an image of the nares reflected.

Although from an early period many attempts were doubtless made to explore the nasal passages, it was not until the value of the laryngoscope in medical work was established that the early laryngoscopists turned their attention to the nose. Foremost of these was Czermak, and a paper of his in the *Wiener Medizinische Wochenschrift* of August 9th, 1859, may be regarded as the first chapter in the history of rhinoscopy. He was at once followed by Türck, Semeleder, Voltolini, Stoerk, and other ardent workers, and the subsequent story of the art has been one of continual progress.

The theory involved in rhinoscopy is the same as in laryngoscopy, the only difference being that the faucial mirror is so held as to reflect the light into another cavity of which an image is accordingly obtained. The apparatus required is also the same. On these points, therefore, the reader is referred to the article on "Diseases of the Larynx." But as the art of rhinoscopy is more difficult than laryngoscopy, it may be well to indicate some variations and hindrances.

In the first place, to explore the nares it is best to select a small mirror—from one-half to five-eighths of an inch in diameter. It is convenient to have the stem curved a little upwards corresponding with the arch of the tongue. An oval mirror is sometimes useful. Some writers direct the mirror to be fixed to the stem at a right angle. This is not necessary, for, as taught by me from 1860, a view of the posterior nares may be obtained by simply turning upwards the face of an ordinary laryngeal mirror. As a rule it may facilitate the examination to have the angle a little less obtuse than for laryngoscopy, but a slight bending of the stem will do all that is necessary, and very great variations can be obtained by depressing or raising the handle.

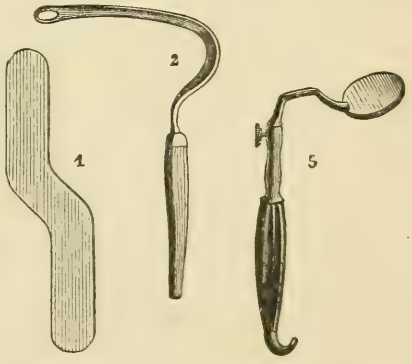

FIG. 1.—Tongue Depressors. 1, Bayonet shape; 2, Türck's; 3, B. Fränkel's.

Seated in front of the patient just as for laryngoscopy, the observer uses the tongue depressor with one hand and introduces the mirror with the other. The tongue depressor may eventually be discarded, but at first is generally indispensable. In trying to assist, the patient often only makes difficulties, since the position of

perfect rest is that in which we want the velum. Combinations of mirrors and depressors have been constructed but are apt to prove only hindrances. The patient can easily himself hold a suitable depressor such as Türck's or the writer's, and then the physician will have both hands free. The greater the space between the posterior wall of the pharynx and the velum the easier it will be to illuminate the nares. When this space is excessively narrow, it may be impossible to obtain a satisfactory view of the parts.

On looking into the mouth, the soft palate may not infrequently be seen to be closely applied to the pharynx, instead of hanging freely so as to permit the mirror to be passed behind it. This is caused by the patient breathing through the mouth, and he must be told to breathe through the nose when the palate will fall forward. Some patients find considerable difficulty in maintaining nasal respiration while the mouth is open, but the ability to do so may be acquired. They may be requested to emit nasal sounds or to take short, rapid respirations. At the moment the current of air is thus sent through the nostrils we may obtain a glimpse of the nasal image, but it is often difficult to obtain a satisfactory view until the patient has been trained or the parts anæsthetized. By one or other plan the most unpromising patient may be enabled to tolerate the examination, provided the conformation of the parts be favorable. But if there should not be space for the smallest mirror between the pharyngeal wall and the velum they may be separated mechanically. For this purpose various palate hooks have been designed. The simplest are best. A rather broad, flat, or fenestrated hook curved at a suitable angle is taken in the left hand, passed behind the soft palate and gently but firmly drawn forwards. Instead of a hook a ligature passed round the uvula to hold it forward has been sometimes used and may be attached by a turn round one of the patient's teeth, to liberate when necessary the operator's hand. A simple twitch may be made by passing a ligature through a cannula, so as to leave a projecting loop. Tape, ribbon, silk, and other substances have also been passed backwards through the nostrils, brought out of the mouth, and secured by tying in front of the lip. These measures are more suitable for operative procedures than for exploration and have been largely displaced by local anæsthesia.

Other obstacles to the introduction of the mirror are enlarged tonsils, adhesion of the palate to the pharynx, and adenoid or other growths. The way to meet these difficulties is to remove the disease. There is one obstacle beyond remedy, viz., when the hard palate stretches so far back as to leave too little available space for the smallest mirror. This conformation is, however, very rare.

The mirror has to be held in such a position as to illuminate the posterior nares. The rays of light must therefore fall upon it in such a manner as to be reflected upon the parts, the image of which is to be reflected on its surface. The plane of this surface will form with the plane of the horizon an angle of about 130 degrees. To illuminate the mirror held in that position, the rays must be directed to a point lower in the fauces than in the practice of laryngoscopy; hence some advise that the patients should occupy a somewhat lower seat than the physician. This will sometimes be an assistance. The patient should sit quite upright, though some advise otherwise; but in the erect position he can easily incline the head slightly either forwards or backwards.

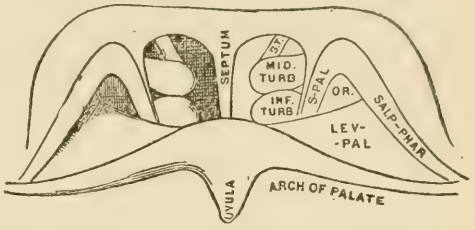

FIG. 2.—Diagrammatic Representation of the Posterior Rhinoscopic Image.

The Image.—What is called the rhinoscopic image is really the combination of several views, each of which is separately examined by the observer. The mirror is held first on one side and then on the other. In each case it is moved more freely than in laryngoscopy, so as to examine as wide a field as possible, and then the two halves—each composed of the several views thus obtained—are blended into one in the observer's mind. It is easy, therefore, to understand that exactness is only to be attained by the utmost care, and all the observer's knowledge and attention are required to appreciate the various views revealed in rhinoscopy, and deduce from them correct indications. At the same time, though this is the most common mode of proceeding, there are cases in which a view of a considerable portion of each half may be obtained in the same image. All drawings of the rhinoscopic image must therefore be more or less diagrammatic, and the greater the extent of surface included in the view the more will this be the case. The accompanying diagram (Fig. 2) shows the position of the several parts and their relations to each

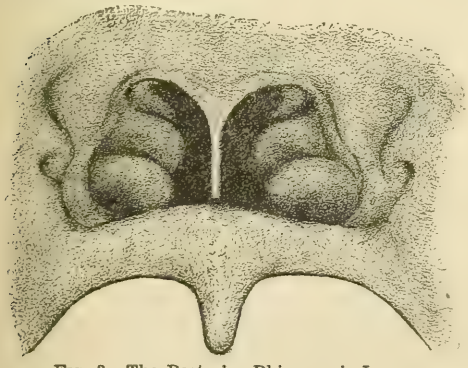

FIG. 3.—The Posterior Rhinoscopic Image.

other. These outlines of the parts should be carefully compared with the details of the image as depicted in the more finished engraving shown in Fig. 3.

The *septum nasi* divides the rhinoscopic image into two halves, one surface of which should be clearly made out on each side. It forms a shining central ridge, narrow below, but increasing in breadth above. At its lowest narrow part, if well illuminated, it may look quite white and bright from the bone shining through the thin mucous membrane. If the light be less brilliant or the membrane not quite so thin and tense, the hue may be pale pinkish, or there may be a yellowish tinge. The color becomes deeper as the septum extends upwards, until at the highest, broadest part it is lost in the red of the mucous membrane of the surrounding parts. The septum seldom occupies the exact median line, but leans a little to one side or other, so that we rarely see a rhinoscopic image precisely symmetrical.

Close to the septum appear two dark, somewhat oval-shaped openings, one on each side. These are the *posterior nares* or *choanæ*, and on careful examination are seen to be partly filled up by three bulbous-looking prominences, of which the middle one is the most distinct. These are the turbinated bodies, formed by the bones of that name and the soft tissues covering them; the *middle* turbinated bodies are easily found, being very prominent and occupying a large portion of the image on each side of the septum. They are covered with thin mucous membrane of a pale pinkish hue, and have been mistaken by beginners for nasal polypi. Just below, and much resembling them, but more uneven on the surface, at the base of the *fossæ nasi*, are two other, somewhat smaller, roundish projections, looking like a pair of almost solid tumors. They are the *inferior* turbinated bodies. They do not seem to approach so near to the septum as the middle ones, and moreover look more solid and duller in color, no doubt partly owing to their being less brilliantly illuminated. Above they overlap the middle bodies, concealing part of their lower borders, while below their own inferior borders are generally hidden by the velum. The *superior* turbinated body usually is reflected only as a narrow projection, in shape almost like a triangle, with its apex pointing downwards, inwards, and somewhat backwards, generally appearing to be lost behind the middle bone. Voltolini found that the posterior extremity of the inferior turbinated bodies swelled up as remarkably as the anterior by the distention of the erectile tissue over the bone. By mechanical irritation they may be caused to swell so as to reach the septum, looking like a pair of tumors, and having a bluish tinge. The middle body is also capable of this erectile change.

Of the three passages between the turbinated bodies the superior meatus is the largest, but only a portion of it comes into the view, and very often it is indicated only by a dark line above the middle bone. The middle meatus is the easiest to discover, and appears toward the outer wall of the fossa. The inferior meatus is very seldom visible. Just outside and behind the inferior turbinated body, sometimes appearing to be on a level with the middle meatus, but really on a different plane, is the irregularly triangular orifice of the Eustachian tube (OR, Fig. 2). The Eustachian openings, often large enough to admit the tip of the little finger, look downwards and inwards, and are rendered more conspicuous by the yellowish color of the circumference.

Below the nasal fossæ it is easy to make out the posterior surface of the uvula and velum—indeed, the beginner may find these parts too apt to come in his way. Turning the face of the mirror upwards so as to bring the reflecting surface into a horizontal position, a view of the vault or roof of the pharynx appears, but this is beyond the scope of the present article.

Practice of Rhinoscopy.—The easiest plan is to pass a small mirror between the uvula and the anterior pillar on one side, and carry it gradually behind the uvula toward the median line. It is then to be withdrawn, taken in the other hand and introduced in the same manner on the opposite side. In this way we may examine each half of the rhinoscopic image and mentally combine the two lateral views into one. In pursuing this method the first point reflected in the mirror is the posterior surface of the uvula, closely following which, or appearing at the same instant, may be traced the posterior surface of the arches and velum, and perhaps one of the teeth will appear. The mirror should now be passed a little higher up behind the velum, which is followed by the eye as it spreads itself out until at the upper part it seems to finish in a sort of fleshy ledge, just above and behind which the septum nasi and the nares come into view. Starting, then, in what may be called the second stage of his exploration, from the septum nasi, the observer should trace this throughout its whole length. On either side of the septum, at the broad upper part may be discerned the nasal opening. This may be traced downwards to the red fleshy ledge already mentioned as formed by the velum, and which intercepts the view of the lowest portion of the opening. The nasal opening having thus been made out, we may proceed to trace its outer boundary, which will be found to be formed by the projecting outlines of the turbinated bones. The most prominent of these is the middle, which appears at its lower border as if the inferior bone overlapped it. Just above this overlapping or upper part of the

inferior bone is the middle meatus, and upon the outer side of this the Eustachian orifice.

The reverse order may, of course, be adopted, and sometimes it will be found easier to begin with the Eustachian tube, and trace the other structures between it and the septum. It is desirable to be able to start from any point, and follow the outline of all the parts as they come successively into view. The general outline of the image must, of course, be familiar to the eye, and then we may begin at any point and pass toward any other without confusion, just as when looking at a map of a locality we know, we glance from one spot to another without losing our consciousness of their relative positions.

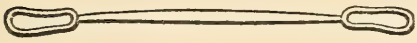

Fig. 4.—Nasal Retractor.

Anterior Rhinoscopy.—The patient, being seated before the observer, may be directed to incline his head so far backwards that the light is easily reflected into either nostril. If now the tip of the nose be raised by the observer's thumb the interior of the vestibule and a portion of the cavities may be brought into view. But, generally, the sides of the nostril require separating. This may be done with a probe or a simple dilator made of silver wire (Fig. 4). A common hairpin has often been utilized as a substitute. It should be bent to a right angle, about three-quarters of an inch from the closed end. Jurasz recommends a retractor made of such a shape, but also bent at the opposite end in the reverse direction (Fig. 5). A pair of these constructed of stoutish silver wire, about three inches in length, are often very convenient. One can be inserted into each nostril, or both into one, according to circumstances. The disadvantage of these instruments is that they occupy the hands of the operator, and therefore are only available for diagnosis. Baber's self-retaining dilator (Fig. 6) is formed of two hooks suitably bent and united by an elastic band furnished with a buckle to fasten behind the head. To one hook he gives a double curve, so that it may pass over the tip of the nose which it draws rather upwards; the other has a simple angle. The degree of dilatation is easily regulated, and both the operator's hands are free.

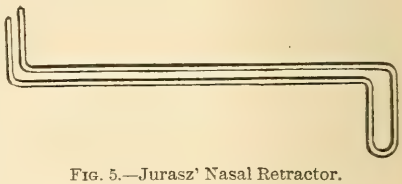

Fig. 5.—Jurasz' Nasal Retractor.

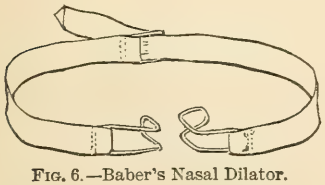

Fig. 6.—Baber's Nasal Dilator.

Markovsky, Meyer, and others employ a bivalve dilator on the

plan of Kramer's ear speculum with a catch to keep it open at any distance. Elsberg and others prefer a trivalve, of which also many varieties are made. Duplay and others again prefer a smaller bivalve consisting of two concave blades which can be separated by a screw. In these it is well to have the septal blade flatter than its opponent.

A very useful and simple instrument is an elongated ear speculum, having either a round or an oval aperture. In fact all ear specula, if made rather longer than usual and with a little flattening on one side, will serve for both purposes.

Fig. 7.—Thudichum's Nasal Speculum.

Thudichum's speculum is very convenient, as it does not require to be held *in situ*, provided that in placing it, it is not introduced so far as to give rise to unnecessary pain. The instrument is composed of two blades connected by a spring. It is sometimes needful by pressure to reduce the power of the spring to a minimum, and occasionally the parts are so tender that this dilator cannot be employed. To meet such cases Messrs. Maw many years ago made for the writer a dilator (Fig. 8) on the principle of the eyelid retractor, in which, by means of a screw, the degree of dilatation can be exactly guaranteed. Fränkel published an article advocating this principle, and so this instrument is commonly sold as his.

Finding that a self-retaining dilator is often a convenience, I

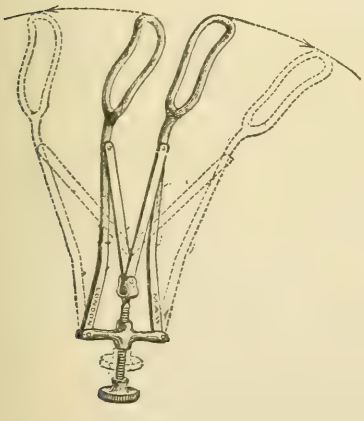

Fig. 8.—Nasal Dilator.

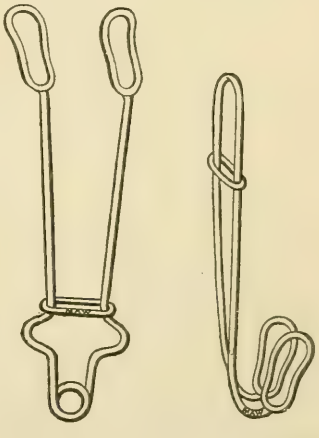

Fig. 9.—The Writer's Self-retaining Nasal Dilators.

afterwards constructed one with similar blades connecting them by a spring, and to prevent too great or painful dilatation I added a ring which enables the operator exactly to control the spring

(Fig. 9). To prevent it being in the way during an operation I have had it bent so as to lie over on the nose when the head is held back.

The Anterior View.—By anterior rhinoscopy we can explore the entire anterior portion of the nasal passage from the superior turbinated body above to the floor below.

The lateral boundaries of the nostrils, especially the inner one formed of the pale-red septum, should be carefully examined. This partition, as we have seen, does not often occupy the median line, the two nostrils are not absolutely symmetrical. Slight irregularities on the surface of the septum are generally observed, and on the upper part there is a bulging into the nasal cavity called the tubercle, which must not be mistaken for a deflection, as it is only a normal formation consisting of an aggregation of glandular structure in the thick mucous membrane.

We may next note the condition of the prominence caused by the middle turbinated body, the anterior surface of which looks toward us, and below it we trace the front and part of the inferior surface of the lower body. The superior turbinated body has scarcely ever been seen from the front, indeed Voltolini denies that it is possible to see it. Just above the middle turbinated body is the roof of the nasal cavity. Below the inferior turbinated body is the inferior meatus, and above the same body the middle meatus. The color of the lining membrane of the anterior nares differs on the different parts. The inferior border of the middle protuberance is normally so pale as to contrast with the rest of the membrane. The septum is of a deeper red, and the inferior turbinated prominence deepest of all, generally a darker red than the mouth, and in contrast with it the middle body looks pale. The appearances may be much disguised by secretion and sometimes it may be necessary to wash out the passages with the syringe or irrigator. In other cases particles may be removed with cotton wool or a brush. The appearance of secretions in this part varies so much that mistakes may easily be made. Another circumstance adds to the variations in the appearances, viz., the greater or less degree of distention of the turbinated bodies. It will be understood that this term is applied to the whole of the protuberances caused by the turbinated bones and the soft structures over them. The anterior extremity of the inferior turbinated body is so largely composed of erectile tissue that the bony support constitutes, according to Voltolini, only about a twentieth part of its bulk. This body in its swollen state assumes a round form, and approaches and sometimes even touches the septum, completely concealing the middle turbinated body and occluding the

inferior meatus. The swollen tissue can be pushed aside by a probe, when it may be observed that the pitting produced is rapidly obliterated. Mechanical irritation such as the contact of a probe or the retractor will often at once cause the body to swell up, and it may be some time before it subsides, so that it is necessary to avoid any such irritant action when we wish to examine the passage. Both distention on the one hand and collapse on the other may also be brought about by nervous influence. Swelling of the part may be set up by irritants suspended in the atmosphere, or by variations in its temperature, as well as in a reflex manner from nervous irritation. No doubt permanent swelling, constituting hypertrophy, must be regarded as pathological and carefully distinguished from the temporary erectile condition, which is normal.

Cocaine is a valuable aid in diagnosis as well as treatment, for its application will cause the swollen body to collapse even as it blanches the mucous membrane of the part, an effect pointed out by me as soon as this anæsthetic was introduced (*Lancet*, November, 1884) and noted about the same time by Dr. Bosworth (*Medical Record*, November 15th, 1884).

In exceptional cases, when the meatus is large and unobstructed, we may see through to the pharynx while practising anterior rhinoscopy; but usually, in order to explore that region from the front, we should have to resort to some such contrivance as Zaufal's tubes, which enable us to examine portions of the posterior wall of the pharynx, but are most useful for the purpose of exploring the Eustachian orifices and their immediate neighborhood. Through these tubes we may also watch the palatal movements, but for studying these Harrison Allen's palato-myograph would be more valuable, as it is both a measuring and a recording instrument.

Anterior rhinoscopy by transmitted light has been employed but not often. The pharynx is illuminated as usual, and the light directed thence into the nares by a rhinoscope held in the usual position. The observer then examines from the front.

Median Rhinoscopy.—Many attempts have been made to explore the central and upper parts of the nasal passage by a small mirror passed along the floor and looking obliquely upwards. A small fenestrated tube with a mirror opposite the opening has also been used, and forms the conchoscope of Wertheim. To prevent the mirror being soiled by the secretion, the opening may be protected by a slide to be retracted by an attached wire thread. No practical results have been obtained by this plan, which has been dignified by the name median rhinoscopy.

Auto-Rhinoscopy.—This term is used sometimes for self-demon-

stration of the parts in the same manner as auto-laryngoscopy in reference to the larynx.

General Rhinal Therapeutics.

The first step in treatment is very often a thorough cleansing of the passages from accumulated or adherent secretions. The method of doing this may vary with the position, quantity, and quality of the accumulation. Moist flakes recently deposited can be wiped off with a brush or absorbent cotton, or they may be washed away with a syringe, douche, or spray. Tough, hardened, adherent secretions may require repeated douches or sprays and sometimes may have to be mechanically detached.

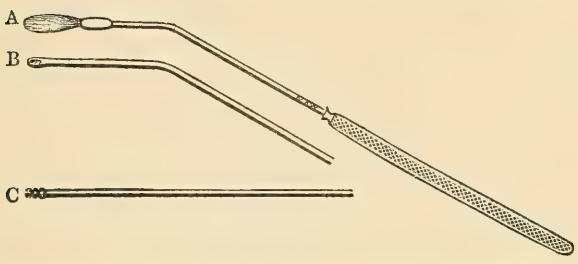

Fig. 10.—Anterior Nasal Brush (A), Probe (B), and Cotton-Holder (C).

Ordinary laryngeal brushes may be easily adapted for use in the nares by bending the flexible part of the stem to a suitable angle. Either straight or slightly curved instruments are best adapted for the anterior nares. The brush and probe are convenient of the shape A or B (Fig. 10), but the cotton holder should be straight. For the posterior nares the instruments should either have a catheter curve or be bent to a right or even more acute angle as shown in Fig. 11.

These instruments may be attached to a shifting or permanent handle and are used not only to remove secretions but to convey remedies to the parts.

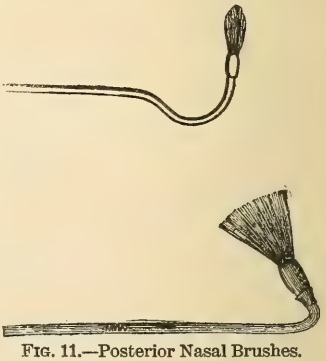
Fig. 11.—Posterior Nasal Brushes.

The *nasal douche* is also employed for cleansing as well as for the therapeutic effects of fluids on the membrane. The simplest apparatus is the ordinary siphon douche consisting of elastic tubing with a nosepiece, the distal end being weighted and placed in a tumbler or other vessel of the liquid (Fig. 12, *a*). Some prefer a special reservoir in the form of a bottle (Fig. 12, *b*) to stand on a shelf or an elastic bag which can be hung up. The latter is very convenient in travelling. In using the douche the patient sits with his head bent forward over

a basin and his mouth open. The nose-piece is passed into one nostril and the fluid flows out of the other. The douche should invariably be used tepid at first. Occasionally the temperature may be reduced after a time, especially when astringents are required. Then

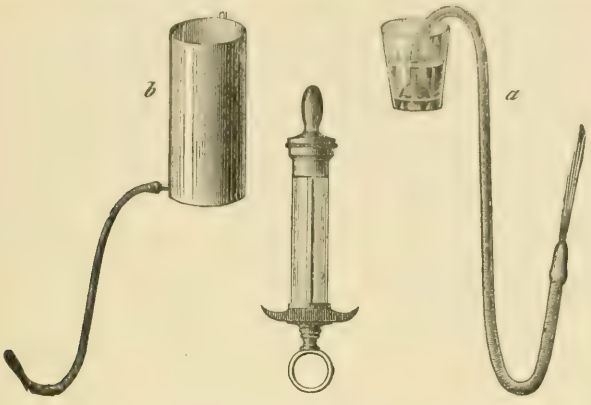

Fig. 12.—Nasal Douches and Anterior Nasal Syringe.

a temperature of 85° F. may be employed and gradually reduced to 75° or even 70°—very rarely lower. The patient should breathe quietly and restrain any movement of deglutition in order that the Eustachian tubes may remain closed; for the accidental entrance of fluid into the ear has sometimes given rise to serious consequences. It is not necessary to have much pressure—just enough to cause the fluid to flow through the nares will suffice, and this will be produced when the vessel is not raised above the patient's head. He should therefore be cautioned not to raise it higher or he may be told to let it rest on his head.

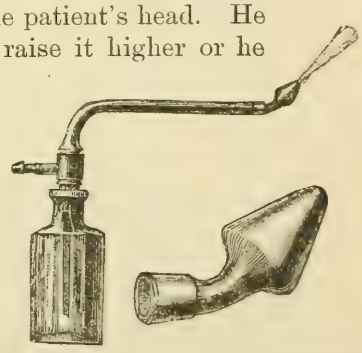

Fig. 13.—Lefferts' Nasal Spray Producer.

The most serviceable liquids for use in the douche are weak saline and alkaline solutions. Half a drachm of sodium carbonate in a pint of water, with a little glycerin added, is one of the best forms for removing hardened secretions. This is less irritant than the salt solution so often used and less likely to injure the ear. Borax or boracic acid may be used for the same purpose. Detergents like muriate of ammonia are sometimes useful. Antiseptics may be added or used alone. The permanganates have a special action on the membrane. Chlorinated solutions, carbolic acid, the carbolates, eucalyptus, and other disinfectants are all useful. It should be re-

membered that a weak saline solution produces less stuffiness than pure water.

In using astringents care should be taken to make them weak enough. Half a grain per ounce of sulphate or acetate of zinc is quite enough. One grain, occasionally two, of sulpho-carbolate will suffice; stronger astringents are apt to give rise to headache, local irritation, and other unpleasantness. Alum is said to have impaired the sense of smell. The douche should be used daily, occasionally twice daily is desirable, and it is sometimes necessary to continue it for a considerable time. Instead of the douche we may sometimes avail ourselves of the primitive plan of snuffing up the liquid from the hollow of the hand. Some persons become very expert at this drinking through the nose, as they call it.

Syringes may be used to detach impacted secretions. Separate tubes are necessary for the anterior and posterior nares. To syringe the latter is very unpleasant to the patient and requires care on the part of the operator.

Irrigators, which the writer introduced many years ago, are preferred by him; they consist of silver tubes of suitable shape perforated at the end by numerous minute holes, so that the liquid can be projected in a fine shower by means of a small Higgenson's apparatus. In this way the passages can be carefully irrigated both from the front and from the back. Old and closely adherent accumulations give way to this irrigation, which, although unpleasant and not to be resorted to in mild cases, is to be preferred to mechanical removal. It is convenient to have the irrigating tubes furnished with a screw to fit the syringe.

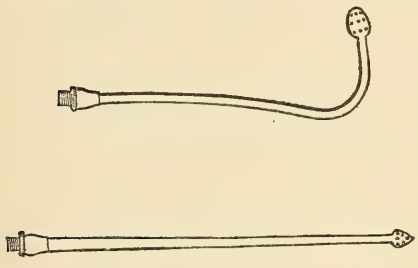

Fig. 14.—Nasal Irrigators.

Sprays, though less powerful, are much more generally useful. Various atomizers have been constructed for the purpose—some for the posterior nares, others for spraying through the nostrils. To separate and wash away accumulated secretions a *coarse* spray will suffice in many cases, perhaps half; for medicating the passages a fine spray is to be preferred. For the former the shape adopted by Professor Lefferts is the best (Fig. 13). It throws a very coarse spray into the anterior nares. The conical end closes one nostril; the fluid is thrown along that nasal passage, and passes out by the other. About an ounce of fluid thus used will generally suffice to cleanse the nares, and so prepare for a fine spray or other remedial

measure. The liquids employed in sprays may be the same as recommended for douches, but the solutions may be a little stronger.

Fumigations and inhalations of course affect the nasal passages but they are most frequently employed for their effects on the whole of the respiratory tract. The inhalation of steam is of special value in acute catarrh and can be carried out in the most simple way by a sponge dipped in hot water. Anodynes may often be added to the water with advantage. The fumes or vapors of various drugs may also be diffused through the room by various methods so as to be continuously applied to the membrane.

Insufflations.—Instead of liquids or vapors powders are often used. Such insufflators as are used for the larynx may be made to serve, but the instrument devised by the writer is more convenient. It is constructed on the plan of the familiar laboratory wash-bottle—the glass tube terminating in proper-shaped endings for anterior and posterior use (Fig. 15). The air tube is bent over before the point of attachment of the rubber tube; this prevents the kinking and the failure to project the powder on compressing the ball which otherwise is continually occurring.

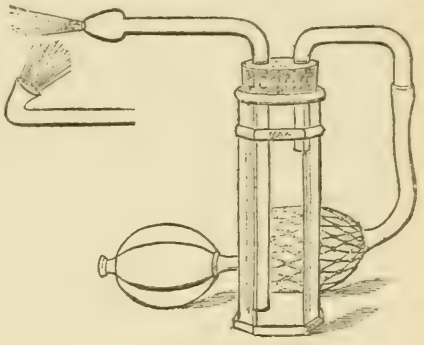

FIG. 15.—Nasal Insufflator.

The powders most suitable for insufflation are tannin, boric acid, iodoform or one of its substitutes, zinc oxide, bismuth carbonate, and occasionally morphine. Salicylic acid is very irritating, but sometimes useful. For diluting the medicaments to a suitable strength starch or powdered acacia is convenient. Sometimes an alkaline powder such as magnesia or carbonate of sodium may be used. The carbolates are also useful in this form. Insufflations are most useful, perhaps, in chronic rhinitis when the mucous membrane is particularly irritable, the secretion rather abundant, tenacious, but not adherent, and the parts sodden and full. They are of little use when there is hypertrophy. In atrophy and ozæna they should generally be avoided.

Snuffs.—Fine powders are easily taken as snuffs, and as the patient can carry them about, the application can be frequently renewed. Anodynes and absorbents suit when liquids prove irritating. Mild astringents suit in other cases. Antiseptic powders may advantageously be used as ingredients of most snuffs.

Nasal bougies, either of gum elastic or soft vulcanite, are very use-

ful. They correspond with œsophageal bougies from Nos. 1 to 9, but are only about eight inches long. Sometimes useful for diagnosis, they are much more valuable for treatment. They may be employed for their mechanical effects, and may also be made purveyors of medicaments, *e.g.*, ointment of iodoform, mercury, etc. Catti's *soluble* bougies are intended for medication of the passages. They are tapering in form, only about three inches long, and one-quarter inch in diameter at the thick end; they are made of gelatin and glycerin, with a little water, the medicament being stirred in when warm, and then the compound poured into a mould to cool. They are easily introduced and, the nostril being plugged with cotton-wool, they gradually melt, the medicament continuing for some time in contact with the membrane. Carbolic acid, tannin, iodoform, etc., can be applied in this manner. *Hollow* bougies to remain some time in the passage are sometimes used, but the perforation is usually too small, and when such an instrument is wanted a catheter answers the purpose.

Nasal sounds are of the same lengths and sizes as the bougies, but made of soft metal. They are useful when it is necessary to effect dilatation more rapidly than can be done with the softer bougies, but they require care in their use.

Laminaria and *sponge tents*, made of the same size as bougies, are also useful for effecting dilatation.

Dilators of various forms have been devised for overcoming stenosis more rapidly.

Nasal catheters may be of silver or soft vulcanite, of similar form to the sounds.

Caustics have been of late years very freely employed. In the posterior nares a properly curved holder is to be used with the aid of the rhinoscope. In the anterior nares a straight or slightly curved holder or probe will suffice. If solid caustic is to be used a sufficient quantity can be fused on to the end, or on either side; if liquid, a pledget of absorbent cotton is wound on the holder, moistened with the fluid, any excess being pressed out, and then carefully applied. Cocaine should be previously applied in the same way and kept in contact with the part long enough to insure anæsthesia. In the anterior nares a speculum or other shield may be used to protect the sound parts from accident. As soon as the cauterization is accomplished, whether in the anterior or posterior nares, the passages should be freely washed out with an alkaline lotion, so as to prevent the action of the agent from spreading.

Nitrate of silver is sometimes classed as a caustic. It is not adapted for the anterior nares nor for the destruction of hypertrophy.

Chromic acid has of late been the favorite. It is very effectual, but requires the greatest care. It can be used solid, a tiny crystal being very carefully fused on the point of the holder. If too much heated it will be decomposed. A saturated solution can be used on cotton. Nitric acid is preferred by many who regard it as the most efficient caustic and sufficiently manageable. In hypertrophy of the inferior turbinated bones a single application will sometimes suffice to reduce it so much as to restore nasal respiration. Glacial acetic acid is less powerful, and unless very slight destruction of tissue is required, less useful. It is inferior to trichloracetic acid. In the posterior nares I prefer solid caustics, no more being fused on to the point of the holder than is appropriate for a single application, so that no accident can occur from the detachment of a portion.

The *galvano-cautery* is preferred by some to chemical agents but is not without its disadvantages. Perhaps the most important is that referred to elsewhere by the writer, viz., that it seems now and then to be followed by serious neuroses. At any rate he has seen parosmia and anosmia after its employment by skilled operators.

Inflammation.

Acute Rhinitis.

This is familiar enough in the form of a common cold; although this derives its name from the fact that it is so often brought on by exposure to a reduced temperature, it should not be forgotten that heat also seems to give rise to many attacks. Indeed, it has been said that "catching hot" would be as appropriate a phrase as "catching cold." Coryza certainly is often induced by sudden change of temperature in either direction and passing from a cold, damp atmosphere to a warm dry one is not seldom followed by this symptom. But the discomfort of the exposed position is more suggestive to the sufferer than the comfort of his warm room. Wet feet are blamed as a direct cause, and a sympathy between the extremities and nasal membrane has been invoked in explanation; but the equally plausible suggestion is offered that the feet more frequently than other parts of the surface are left for considerable times to the influence of cold and damp. Irritating substances, whether gaseous or solid, will set up acute coryza, which may be distinguished as *traumatic*. The exanthemata are frequently ushered in by coryza, and it is often the earliest symptom of influenza. The rapid spread of this epidemic is due to contagion and the way in which a common cold spreads through a household satisfies many of its infectiousness and suggests

that, like the epidemic, the ordinary form may be due to microbes. But rhinitis may also arise from other causes. It is, for instance, a frequent result of taking iodide of potassium.

It seems unnecessary to describe the symptoms of a cold in detail or to follow it through the early dry or stuffy to the second or moist stage, with its abundant watery acrid discharge succeeded by one of a thicker, muco-purulent but less irritating character, that in turn giving way to a thinner one which gradually diminishes until the normal condition is regained. It may be well, however, to note that extension to the accessory cavities gives rise to special distress, heavy, dull aching in the forehead pointing to implication of the frontal sinuses, severe pain in the cheek to the antrum of Highmore, epiphora to obstruction of the lachrymal duct, noises in the ears and deafness to the same state of the Eustachian tube. These symptoms do not necessarily imply extension of the active inflammation, though that more serious event may occur, but they are brought on by the swelling of the membrane in a state of congestion, the pressure thereby exercised on the nerve endings, or the accumulation of discharges about the orifices of the cavities and tubes named.

Pathology.—Hyperæmia, congestion, and inflammation of the mucous membrane. Quite early lymph corpuscles and epithelial cells rapidly increase, making the flux cloudy and subsequently opaque. The membrane is red and turgid, and perhaps the vessels are visible. Later ecchymoses, abrasions, or small ulcers may appear.

Rhinoscopy.—Anteriorly in the early stage the dark deep-red and swollen but dry and perhaps glazed appearance of the membrane, especially over the turbinated bodies, is most obvious. Later this changes to a more pink hue and the glazing or dryness gives way to the moist shinyness of the second stage. The swelling is then less, perhaps the subsidence permitting an extension of the view. But even later still the swelling will not have decreased to the normal point and the color will still be the deep red of inflammation, though this appearance may be apparently modified by the bright-yellow discharge covering it.

Posteriorly the nares are seen to be more or less blocked by the swollen turbinated bodies, and the congestion can be traced extending to the pharynx and perhaps to the larynx.

Treatment.—The remedies that have been proposed for a cold are innumerable and every member of the community seems to be proud of possessing a "sure cure" and always ready to prescribe it. Here it is only proposed to mention some of the methods the writer has found most useful. And first of all it may be asked can one abort a cold? In numerous cases yes, and for this purpose preparations

of opium and belladonna are the best internal remedies. The time-honored dose of Dover's powder at night followed by a saline purge in the morning is effectual, although rather nauseous. A dose of morphine and atropine is more agreeable and quite as certain if not more so. Personally I have many times arrested a cold in the early stage by a single dose of a ¼ grain of morphine, often by ½ grain. If this last prove insufficient it may be followed by a second ½ two or three hours later. The addition of $\frac{1}{100}$ to $\frac{1}{60}$ grain atropine or an equivalent of a reliable preparation of belladonna reinforces the remedy and prevents some of the unpleasant effects, *e.g.*, headache and constipation. In some cases the belladonna may be advantageously given in divided doses at intervals of two or three hours. As a substitute for atropine in various cases I have tried daturine with satisfaction. Its action seems very similar. I use a one-per-cent. solution of the salicylate. This plan does not produce the sweating which usually follows the Dover's powder, when the patient is kept warm in bed after a foot bath, or supplied with hot drinks and diaphoretics, of which a combination of acetate of ammonia and spirit of nitrous ether is as useful as any. When the sudorific plan is adopted, soothing inhalations may be added with advantage, but should not be continued after the moist stage has set in. The moist heat conveyed by the steam is the main thing in all these inhalations and the simplest way of applying it is the best in these cases. A basin of hot water and a sponge are all that is required. The sponge wrung out of the water, as hot as can be borne, is placed over the nose and mouth. Breathing through it the nasal passages are fomented much as the external nose is. Browache and dyspnœa are both relieved. The water may have a few drops of carbolic acid or other remedy added to it when thought desirable. Many robust people find a Turkish bath will abort a cold. A lamp bath in the bed-room is safer. A few have tried pilocarpine, but it is neither agreeable nor more efficacious than the other plans. Some few patients seem quite unaffected by the morphine which is so prompt to relieve others. In such cases the local action of cocaine is successful. It may be mentioned that the coryza of iodism is easily controlled by morphine. When there is much febrile excitement a few doses of aconite may be given.

Either with or without sudorifics, abstinence from all liquids for twenty-four to forty-eight hours, urged by C. J. B. Williams, following Lower, is still sometimes resorted to by patients, but they mostly declare the remedy worse than the disease. Some persons find a full dose of quinine at the outset of the sudorific treatment useful. I have not seen the benefit of this unless the fever ran so high as to

cause apprehension of something more serious than a cold. It has certainly no effect on the rhinitis, but at a later stage of catarrh repeated small doses are useful.

When treatment has not been begun in time or has failed to arrest the disease in the first stage, we have to deal with increased secretion—the veritable catarrh. Then the most successful plan is to continue small doses of belladonna through the day and repeat the morphine at bedtime. If fever calls for it, the belladonna or atropine can be added to the acetate of ammonia mixture or preferably perhaps an aconite mixture. Astringent sprays and injections have often been recommended but should be resorted to only at a much later stage—in fact, only in cases in which a chronic discharge has continued after other symptoms have passed away. Cocaine in a weak alkaline solution used as a spray is more effectual in these cases than any astringents. Powders are sometimes preferred used as snuffs, and cocaine may be used in this form, so also may morphine. Ferrier dilutes the morphine with gum arabic and nitrate of bismuth. Carbonate or oxychloride of bismuth are finer powders; magnesia as an alkaline may be added, and tragacanth has the advantage, if any gum be used, of swelling up more and so causing the powder to adhere more closely to the membrane. A little menthol, boric acid, or other medication can be added. Camphor is still used in this and other ways, but seems to have nearly lost its once great popularity.

Michel recommends silver nitrate in snuffs but I have not tried it. Bosworth, after the vascular plethora and serous exudation have been brought under control, advises making a small eschar with chromic acid on the lower turbinated body "to pin down the swollen membrane and prevent the return of blood to the part." He admits the need of exceeding care to have the tissues thoroughly contracted by cocaine before applying the caustic, as if the measure fails it "will have done more harm than good." I have never resorted to cauterization in so early a stage. Possibly in persons so sensitive as to be subject to rhinitis on the least provocation, such a plan might be worth a trial, though the mode of action may be only hypothetical.

When the accessory cavities are threatened, fomentations as hot as can be borne should be resorted to at once and kept up for hours. The large sponge is convenient for applying the heat and affords the means of inhalation at the same time. Hot douches or injections into the nares are sometimes preferred and if used should be made antiseptic. At the same time morphine and atropine should be pushed internally, and often cocaine locally with a view of preventing the necessity of more severe measures.

Acute Rhinitis in Infancy

is a graver disease, often attended with severe dyspnœa which may assume a paroxysmal form; it has some resemblance to laryngismus stridulus or to croup, and even when there is not unusual dyspnœa the act of suckling is interfered with so that a cold necessitates feeding the baby for a time with the milk drawn from the mother, and in some cases weaning it altogether. The reason is that the nasal passages are very small in infancy—even relatively as well as absolutely small. This is most obvious in the middle meatus, of which the anterior opening is quite minute, and the free extremity of the inferior turbinated bone is longer and more curved than in the adult (Kohts and Lorent), leaving a very narrow passage which increases in size with the general growth. Further, during sleep the infant's tongue falls back against the hard palate, and even when the lips are not closed breathing has to be carried on through the nose; rhinitis causes swelling of the mucous membrane, and a very slight degree of this must in such circumstances interfere with respiration. Hence the dyspnœa, paroxysms of which are prone to occur as soon as the infant falls asleep. Hence, too, the difficulty of suckling while the swelling of the membrane continues.

In regard to treatment opiates are of course inadmissible. The best plan is, in the early stage, to arrange a croup tent and later to keep the infant in a warm, dry atmosphere, taking care that it has sufficient nourishment. A small soft tube may be passed through the nose while the infant is suckling, but this will seldom be required. In an extreme case milk could be administered through a tube.

Purulent Rhinitis.

The inflammation in catarrhal rhinitis may be so violent as to precipitate the formation of pus almost from the outset. This, however, is a very rare event and when it occurs is accompanied by high fever. A purulent discharge is more frequently the result of traumatism. It may be set up by the inhalation of irritants such as ammonia, bromine, chlorine, etc. It may also result from operations in the nose whether by cutting or cautery. Purulent rhinitis is also seen sometimes in the newly born infant and is usually attributed to infection by the maternal discharges just as in the case of ophthalmia neonatorum. Some of these cases may, however, be produced by soap or other irritating substances or rough usage in the first washing of the child. Gonorrhœa affecting the mother may no doubt be conveyed

to the infant at birth. This form of rhinitis has also been seen in adults, mostly through infection by the patient's own finger or handkerchief, and cases have been recorded in which soiled linen has conveyed the disease to another person. A muco-purulent discharge is also met with in older children, which appears to follow repeated colds and when established becomes a very chronic and obstinate disease. Bosworth seems to regard this as really the first stage of ozæna. There is not here the swelling that in acuter cases interferes with nasal breathing, though the accumulation of secretion may do so, and of course every fresh cold aggravates this and other symptoms. A purulent nasal discharge is found in ulceration of the membrane, however produced, and is seen in the rhinitis of diphtheria and the exanthemata. Foreign bodies lodged in the nares also give rise to a purulent discharge.

Diagnosis is founded on the purulent character of the discharge, but it is important to distinguish the several forms and the history will often enable us to do this. In the newly born the possibility of infection is not likely to be overlooked, and the extension to the conjunctiva is significant. At a later date the history points to a succession of catarrhal attacks having gradually established the condition of chronic suppuration involving both nostrils, in contradistinction to a foreign body which generally affects only one side. These bodies introduced by children are so often forgotten that a one-sided discharge should always suggest careful search.

Rhinoscopy reveals the turgid, swollen, or sodden state of the membrane according to the stage, but the surface is veiled by the discharge, which must therefore be removed by sprays or douches before the exact condition of the membrane can be determined.

Treatment consists in thorough cleansing of the passages and keeping them as free as possible from accumulations of discharge. This is best accomplished by tepid, alkaline, and antiseptic sprays, and perhaps at a later period a weak astringent may be called for. In the case of infants it may sometimes be desirable to employ a temporary post-nasal tampon while syringing or spraying, in order to prevent the fluid from gaining access to the larynx.

Membranous Rhinitis.

Instead of pus, acute inflammation sometimes leads to the formation of a fibrinous exudation which is deposited as a false membrane. Some observers regard this as always diphtherial. It is accompanied by fever, the temperature at first being 101° to 103°, but, except in children, falling again on the second or third day to 100° and perhaps

to the normal. A membranous rhinitis is often set up by operations, especially by the galvano-cautery. The disease begins as an acute rhinitis or severe cold with pyrexia, but the first stage is very brief or quite absent, a discharge of a sero-mucous or sero-purulent character coming on early and continuing throughout. Nasal respiration is impeded by the exudation and by the swelling of the pituitary membrane, and frontal headache is often distressing. Pain in the course of the nasal nerve is common and anosmia is general.

The *diagnosis* is founded on the presence of the false membrane which is easily detached without causing hemorrhage. This membrane, unless it comes near the entrance of the nostril, will probably not be detected until the passages are cleansed from secretions, and it may be hidden by the swelling of the turbinated bodies and only proved to be present by obtaining a portion on a brush or a pledget of cotton. Cases arising from traumatism are not difficult to explain. Such as seem to arise spontaneously suggest a relation to diphtheria, and a proportion of the recorded cases are almost certainly examples of that disease, which will be treated at length in a later volume of this work. Microscopically the membrane precisely resembles that of other croupous deposits.

The *prognosis* is favorable, the membrane seldom spreads, but comes off in shreds and may be renewed in four or five days, the disease lasting two or three weeks in adults but extending to several weeks in children.

Treatment is to be directed to the removal of the accumulation by antiseptic alkaline sprays followed by an astringent. Moldenhauer advises inhalations, Bresgen and Hartmann iodoform insufflations, Bosworth painting with tincture of iron. A fresh solution of recrystallized chloride of iron would be better as avoiding the action of the free acid of the tincture.

Chronic Rhinitis.

This exhibits in a less intense degree the train of symptoms which characterizes the acute form. In fact, the chronic inflammation commonly originates in an acute attack, which instead of passing rapidly into complete resolution gradually fades through a subacute into a chronic state. This is more likely to follow a series of repeated catarrhs than a single inflammation of more violent type. It may also be induced by constant or frequent exposure to any of the causes of inflammation. Thus many snuff-takers have a constant slight catarrh, and other irritants may well give rise to a farther degree of the disease. Snuff taking and smoking have been freely

blamed as causes of chronic rhinitis and are generally looked upon as aggravating the condition or rendering it more intractable. Tobacco must certainly be regarded as an irritant, but at the same time it is equally obvious that the membrane becomes accustomed to the stimulant. No age is exempt, but children whose delicate membrane is very susceptible to exposure are specially liable to the disease. In the aged a slight increase of secretion is sometimes classed as nasal catarrh, but in these subjects the liability to repeated colds passing on to bronchitis masks the minor ailment. There is generally increased secretion—one may say always at some stage—and this may be excessive and constitute the most prominent symptom. In other cases the patients complain most of stuffiness, and there may be sufficient swelling to cause considerable obstruction in the passages, when the vocal change erroneously called "speaking through the nose" will be present. The sense of smell is more or less impaired.

Diagnosis.—The characteristic color of the membrane is seen in the rhinoscope when the parts have been cleansed. Otherwise the surface is often covered by secretion which, if rather thick, is apt to appear in patches. Erosions or ulcerations are sometimes seen, most frequently in the vestibule; if on the septum they may cause perforation. In other cases the feature which overshadows all others is the thickening of the membrane, particularly over the turbinated bodies. When there is excessive thin discharge, the presence of a polypus may be suspected, and in all cases it is desirable to make sure of the absence of any new growth in either the nasal passages or the accessory cavities. When rhinitis is due to syphilis the deeper tissues are commonly involved.

Pathology.—The morbid changes vary somewhat. Proliferation of epithelial cells and infiltration of sub-epithelial tissue are the most constant. The membrane is thus generally thickened and its glandules more or less injured by pressure from the indurated tissues around. The swelling caused by the venous distention is most marked over the turbinated bodies.

Treatment.—If there is much irritation, sedative insufflations may be used at first, the patient being cautioned not to aggravate the condition by continually blowing the nose. A little eucalyptus oil used on the handkerchief as a perfume is agreeable to many. If there is not excessive irritation, weak astringent sprays should at once be employed. A spray containing sulpho-carbolate of zinc or acetate of zinc gr. ij. and glycerin of carbolic acid ♏ij. to the ounce is astringent and the carbolic acid in such dilution is rather soothing. When the discharge is thick and tenacious it is well to begin with alkaline lotions. Five to ten grains of carbonate of soda or borax

in one ounce of water with a little glycerin added will meet the indication. A grain of carbolic acid may be added with advantage. As soon as the discharge has become thin, astringent sprays should be substituted. In some very obstinate cases I have employed soluble bougies containing iodoform or iodol, leaving them to dissolve in the passage.

The disease may be regarded and treated as local, but of course when any dyscrasia is present, general remedies should also be prescribed. Questions of diet, regimen, change of air and climate may thus arise and have to be determined by the constitutional rather than the local state.

Hypertrophy and Atrophy.

These conditions of the pituitary membrane and their relations to each other have for some years engrossed much attention. They constitute two very different varieties of chronic rhinitis. Indeed, the one set of cases affords a striking contrast to the other. In hypertrophy of the membrane the lumen of the passages is narrowed and obstruction to the nasal respiration is the leading feature. In atrophy there can be no obstruction as the passages are widened by the process. Many rhinologists, perhaps most, have adopted the suggestion that they are but different stages of chronic rhinitis, the membrane becoming hypertrophied as a consequence of long-standing inflammation and at a still later date atrophy setting in. Clinton Wagner speaks of atrophy as "merely a later stage;" Mackenzie says it "appears to be always a secondary affection;" Lefferts speaks of it as a result of hypertrophy or as "more rarely occurring early in the disease before hypertrophy is far advanced;" Shech says in the majority of cases it is preceded by the hypertrophic stage; Schäffer speaks of hypertrophy lasting eight or ten years before atrophy is induced. On the other hand it is held by some that the two diseases are distinct. Seiler thinks "it may be a sequence to the hypertrophic stage or may be of the atrophic variety from the start;" Sajous says atrophy "ocurs as an occasional result of hypertrophy." At the International Medical Congress in London (1881) Dr. Bosworth announced that, although he had previously taught that atrophy was a result of hypertrophy, he had abandoned that view and believed the two affections to be different "from the outset;" he held that it commences as a desquamative inflammation which develops in the direction of glandular atrophy and leads to destruction of the muciparous glands and follicles, seeming to regard purulent rhinitis as its early stage. In a process taking so long to complete,

it is obvious that few opportunities of watching a case through all the stages can be expected, but Schäffer says he has followed cases through the hypertrophic to the atrophic stage, and Clinton Wagner reports a case in which he saw this change effected between the spring and autumn of 1881. Mackenzie says he has "occasionally seen the atrophic condition reached in the course of a few months." Interesting as the relations of the two conditions are, they present such different aspects and require such different treatment that it is convenient to consider them separately.

Hypertrophic Rhinitis.

This presents the features of chronic catarrh with more or less impediment to nasal respiration according to the extent of thickening that has taken place. Many patients when they first apply for relief are constant mouth-breathers. Most of them can breathe through the nose, but it costs them an effort. This is specially noticeable in children. The voice has also the characteristic nasal twang. The adult will complain of constant and increasing stuffiness and perhaps say he is quite unable to blow his nose. There is diminished sensibility of the membrane, and smell is impaired or lost. The secretion is thick and tenacious but not offensive. It is commonly rather excessive, or at least seems so to the patient on account of his desire to get rid of it, but it may be really scanty. Some of it in passing toward the fauces, on account of its unnaturally thickened consistence, sets up hawking and perhaps coughing. The accumulation of the night is not easily got rid of in the morning and the effort not seldom brings on retching or vomiting. Post-nasal and pharyngeal catarrh are frequent complications, modifying but increasing the cough, which is apt to be paroxysmal should the uvula be long.

Etiology.—The continuance of chronic inflammation for a considerable period would seem a sufficient cause for hypertrophy of the nasal membrane as of other tissues. Such chronic inflammation frequently succeeds acute attacks. Nasal catarrh is too common an occurrence to be easily excluded, and most patients give a history of frequent colds. It has, however, been suggested that the disease is a distinct variety from the beginning and that the numerous colds are in reality exacerbations of the chronic inflammation. A good deal has been made of climatic conditions, for the nasal passages are peculiarly exposed to the influence of atmospheric changes. Hypertrophic rhinitis is very prevalent in America, and it has been suggested that something in the climate may account for this prevalence. If so it should be something that also exists in the climate of London

where the disease is likewise common. Possibly the enthusiasm with which American physicians have studied nasal diseases may have given rise to the common impression. Beverley Robinson finds trades in which irritating vapors or dust are more or less constantly inhaled an efficient cause. This surmise is not, I think, supported by statistics. Various constitutional states have been called in to account for the disease, but it is constantly seen in patients where there is no dyscrasia. The old idea of a catarrhal diathesis seems to have died out. Any impediment to nasal breathing is said to be a potent cause of hypertrophy. Hence the importance attached to deflections of the septum and other deformities. Injuries, too, may give rise to this condition, and it has been surmised that falls on the face in infancy are not seldom the starting-point of chronic rhinitis. This may well be in cases where the accident has produced distinct deformity, but if the ordinary tumbling of children learning to walk could set up hypertrophy, nasal obstruction would be universal.

Pathology.—The morbid appearances found are those met with in chronic inflammation of mucous membranes elsewhere, with such differences as are due to modifications of the structure to adapt it to its special function in this locality. The membrane is thickened throughout. The epithelial layer is greatly increased, but there is no tendency to free desquamation. Some of the cells are often found in a state of fatty degeneration. The adenoid layer is likewise thickened, but here connective tissue has more or less replaced the adenoid elements. The capillaries are large and full and the acinous mucous glands very numerous. In the submucous layer again there is great increase of connective tissue, especially between the numerous enlarged veins. In some specimens there is a myxomatous change, especially over the anterior end of the middle turbinated bone, so that in appearance and density there is considerable resemblance to a polypus, which formation is in fact often accompanied by hypertrophy.

Diagnosis.—Anterior rhinoscopy shows the membrane to be rather hyperæmic, swollen, and perhaps coated with a thick muco-purulent secretion. The color is dusky red, derived from the capillaries of the mucosa, not dark as in purely venous congestion, nor bright red as in acute rhinitis. But the swelling is the characteristic appearance. When of moderate extent we may trace its congested, irregular surface on the lower and chief part of the middle turbinated bodies. On the middle one the color is brighter red. The swelling over both bodies of course restricts the view according to its degree. When this is considerable, the lower appears to project far into the nasal passage and may stretch across and impinge on the septum so as to

cause complete obstruction. In such cases the hypertrophied membrane may hang down loosely and encroach on the inferior nasal meatus. It then presents some resemblance to a polypus, but this growth usually descends from a higher level. Hypertrophy, too, is mostly symmetrical and bilateral. Occasionally the swelling in chronic perichondritis may closely resemble that of hypertrophic rhinitis, of which a striking case was reported by Gottstein. The thickened membrane pits on pressure with a probe and returns to its shape rather slowly. The membrane over the middle bone is never affected to the same extent and sometimes scarcely at all.

Posterior rhinoscopy brings into view the posterior ends of the middle and inferior turbinated bodies. The membrane here is thickened, corrugated, irregular, and pale, almost whitish, forming a characteristic tumor on each side of the septum and closing more or less the posterior nasal opening. The closure is often rendered more complete by a thickening which forms an irregular ovoid tumor upon the side of the septum, as has been well figured by Lefferts. This tissue like that in front pits on pressure with a probe.

The swelling of the membrane may be temporarily reduced by spraying with a four-per-cent. solution of cocaine, when a larger surface will be brought into view and the membrane will appear to fit closer to the tissues below so that perhaps the shape of the bones is more obvious, but never to the extent that would occur in the normal state.

Treatment.—The first step is that which has been pointed out as necessary for complete diagnosis, viz., removal of accumulated secretions which can be accomplished by an alkaline lotion thrown into the passages in a coarse spray. So, also, cocaine will have been used in many cases. This is to be followed up by a weak astringent and antiseptic spray used by the patient two or three times a day. The zinc and carbolic lotion already named will answer the purpose, but other mild astringents may be substituted or used alternately. A solution of iodine gr. i. to ʒ i. with potassic iodide gr. ij. is often effectual. Now and then an ointment containing mercury may be employed. In some cases snuffs may suit better, and they have the advantage of being easily carried about and used without apparatus so that patients will apply them more frequently.

When the hypertrophic process has not proceeded far the above measures may suffice. In other cases soluble bougies may be made the vehicle of medication. When hypertrophy is more decided, gum elastic or flexible metallic bougies as recommended by Clinton Wagner may be resorted to. Here the principle of treatment is different. We are resorting to dilatation. Beginning with small sizes we can

gradually increase. At first a bougie may be left in the nostril for five minutes, and this time can be gradually increased in successive applications; in a week or ten days the bougie can be retained for half an hour, often longer. In inserting the bougies the utmost gentleness is necessary, as only mischief will be done if too much force is employed. The passage may, however, be first treated with cocaine, which will contract the membrane and so let a larger size pass.

In more advanced cases, especially when the hypertrophy is mainly over the inferior turbinated bone, it may be necessary to remove part of the redundant tissue on one or both sides and thus restore the power of nasal respiration. This can be done by either caustics or other surgical measures. In either case it is important for the parts to be thoroughly anæsthetized. The caustic to be preferred is trichloracetic acid, chromic acid, or nitric acid; silver nitrate is useless. The fluids are applied by means of a morsel of absorbent cotton wound on a suitable probe, and great care is to be exercised to restrict the application to the spot it is intended to act upon. Chromic acid is very efficient and easily applied. A crystal should be carefully fused on a glass or silver rod; this is easily done by touching the point of the rod on a drop of mucilage and then on the crystal, and holding it thus charged over the flame of a spirit lamp until the acid fuses. When cold the charge can be seen by the color. Immediately after cauterization the passages should be well washed out with an alkaline lotion. Instead of chemical caustics the galvano-cautery has been extolled by numerous operators, and to those who have occasion to use it constantly no doubt it is very efficient. Others, however, will find it troublesome and it is by no means easy to regulate the degree of heat at the moment of contact with the tissues. I have on a former occasion called attention to one disadvantage of this method, viz., the effect on the sense of smell. Either anosmia or perversion may follow its use; this perversion is sometimes more distressing than the loss of function; it may endure for a long time or perhaps be permanent.

Instead of by caustics, hypertrophied tissue may be removed by the sharp forceps or an écraseur. The former is more severe and scarcely ever need be resorted to. Jarvis' snare is perhaps the best instrument. The loop of this écraseur passed over the tissue to be removed is easily tightened by a few turns of the screw, and by taking time over the operation hemorrhage is reduced to a mere trifle. In operating on the anterior turbinated body it should be transfixed by a curved needle and the loop passed over this so as to prevent slipping. When the posterior extremity of the turbinated body is affected, the loop should be of such size and bent to such a degree before passing

it into the naris that it will slip over the growth, which should then be cut through very slowly so as to prevent hemorrhage. This is seldom severe in these cases, but should it be so injection of hot water will restrain it, and this failing, the nares could be plugged from in front or behind.

Instead of excising the hypertrophied tissue I have had some success from multiple scarification or puncture, and would like to see this plan tried by others. Anæsthesia is not always necessary on account of pain, which is only slight, but it is desirable on account of the contraction caused by cocaine. A very small knife with a cutting edge only at the point is the best instrument. A few fine lines drawn along the hypertrophied tissue will destroy new vessels and induce absorption. This very easy operation can be repeated from time to time. The nostril should afterwards be plugged with iodoformed wool, or a soluble bougie of iodoform can be introduced and retained in place by a cotton plug.

Atrophic Rhinitis.

This affection presents a striking contrast to that last described. Instead of the nasal passages being narrowed by increased thickness of their lining as in the hypertrophic form, they are widened by a wasting of the membrane, and this process may extend to the subjacent tissues, even the bony structures being sometimes involved. Accordingly there is no obstruction to nasal respiration except of a very temporary character from accumulations of inspissated secretion, which are often expelled with difficulty.

Symptoms.—There is the greatest variation in the symptoms. Sometimes scarcely any uneasiness is present, the sensibility of the membrane being apparently diminished. In other cases there is much irritation or itching, provoking the patient to scratch or pick his nose so much as to wound the membrane and bring on ulceration. Perforation of the septum has sometimes been thus induced. Thickened discharge or crusts are from time to time expelled, especially in the morning. When the accumulation of the night is thus got rid of, any feeling of stuffiness is removed, the wide passages being left free. The sense of smell is impaired or lost.

Ozæna is so constant a symptom in atrophic rhinitis that the word is often used as a synonym for that condition. It has, however, been long applied to signify any nasal affection in which there is a marked fœtor. Ulceration of the membrane and diseased bone may cause a very offensive smell, though it is of a different character and cannot be completely removed by cleansing sprays. Malignant

disease, too, is accompanied by fetid discharges. A foreign body in the passages may give rise to a very offensive smell. In atrophic rhinitis the ozæna is of a peculiarly sickening kind and very penetrating, so that it is a source of distress to those surrounding the patient. Odors are difficult to describe. This is so unlike any other that once perceived it will always be recognized. Although ozæna is so constantly associated with atrophy, doubts exist as to the rule being absolutely universal, for one may have ozæna when no atrophy can be detected, but of course it is quite possible for it to be present in some position beyond the range of the rhinoscope. It is much more common, however, to meet with atrophy without ozæna. The source of the odor is generally attributed to decomposition of the secretions, and these cases might be explained by supposing that the discharge was not retained long enough in the passages to decompose. It is difficult, however, to apply this explanation to some of the cases.

Diagnosis.—On examination anteriorly the amplitude of the passages is the most obvious feature, and we can often see through to the posterior wall of the pharynx. The turbinated bodies look diminutive, and adhering to them and perhaps to the septum are grayish or yellowish-brown crusts. These occasionally stretch across the passage and then, of course, interfere with the view. Sometimes there is such an amount of this inspissated secretion as partially to block the passage. When hardened crusts are mechanically removed, the membrane beneath is seen to be congested and it sometimes bleeds. Even when only a spray is employed, which is the best method of cleansing, there is occasionally slight bleeding. Beneath the crusts there is often a muco-purulent secretion. Half an hour after the removal the congestion of the membrane will be much diminished and it may look quite pale and thin. Small erosions or ulcers are occasionally seen but these are due to accidental circumstances. Posterior rhinoscopy shows the turbinated bodies much less affected. Crusts are rare in this part. There may be a few flakes of semi-solid secretion on the surface, but they are not closely adherent. The mucous membrane is moist, thin, and perhaps somewhat pale. Sometimes it looks rather shrunken and perhaps glazed. There is no enlargement of the posterior ends of the turbinated bodies—if anything they are undersized. The pharynx exhibits a dry and glazed mucous membrane, sometimes compared to parchment, but it is of a red or dark-red color, shrivelled and thin, evidently atrophied. In the naso-pharynx there is a similar condition, but the dryness is not so marked and plugs of discolored secretion are often adherent.

Etiology.—Many have thought that there was some constitutional

condition predisposing to the disease, but there seems no reliable evidence that such is the case. Scrofula, tubercle, and syphilis have been blamed, but it is common enough where there is no trace of either. The age of puberty is the period when most cases come under observation, though undoubtedly the process begins earlier. Mention has already been made of the theory that it starts from the purulent rhinitis of childhood. Zaufal suggested congenital deficiency of the turbinated bones and Chatellier rarefying ostitis as causes, but Zuckerkandl found no such condition in the course of an examination of two hundred and fifty-two children's skulls. The rapid drying of the secretions has been attributed to a chemical change in the mucin, to deficiency of ciliated epithelial cells (Cohen), and to a decreased amount of the fluid constituents. Vieussens attributed the ozæna to a fermentative change, and this would explain why the smell should sometimes return so soon after cleansing that there seems scarcely to have been time for decomposition; yet this last process in such a position as the nares might well occur early. Krause attributes the smell to fatty degeneration of the mucous cells, the fatty matter becoming acid. E. Fränkel, however, could find no fat in three cases at post-mortem examination. Michel, like Reininger, thinks suppuration of the accessory cavities may be the cause. Of course there has been an active search for microbes. Loewenberg described a large organism in the deeper strata of the membrane, and is supported by Noquet, but Habermann failed to find it. Thost found diplococci in the secretions. These organisms may perhaps be referred to decomposition of the crusts. Hajek (*Berliner klinische Wochenschrift*, 1888) isolated a short rod from the discharge which when grown upon gelatin at 37° C. set up a powerful odor of putrefaction—Bacillus fœtidus ozænæ. Another organism is Reimann's (Inaugural Dissertation, 1887) Bacillus smaragdinus fœtidus, which is also a little rod, and it gives rise to a very offensive odor. Possibly one or both of these may be the cause of the horrible odor of ozæna.

Unsuccessful attempts have been made to inoculate the disease in the nares of animals. Injected subcutaneously in rabbits the fetid secretion causes inflammation and death.

Pathology.—Much that might be said here has been anticipated. It is natural enough to think of atrophy as succeeding hypertrophy and as to a large extent brought about by the encroachment of the increased connective tissue. In this case, too, the hardening crusts have been considered as additional sources of pressure. On the other hand it is held that the process is one of atrophy from the beginning, that there is no increase of connective tissue but active

epithelial desquamation and a transformation of epithelial cells into inflammatory corpuscles. Gottstein, however, at a post-mortem examination found the epithelium normal, beneath this a layer of round cells with a few of spindle shape, and below this a layer of areolar tissue, the fibrillæ lying parallel to the surface and collected into bundles in different stages of development. The glandulæ were numerous but their contents were hazy and the gland cells misshaped and hardly to be recognized. Krause found the epithelium horny and the mucosa converted into a kind of dense connective tissue, diminution in the number of vessels and thickening of the adventitia of those left, producing more or less obliteration. The glandulæ also were lessened and those that were left were in a state of fatty or granular degeneration.

Prognosis.—The disease is exceedingly intractable. In young children it is sometimes arrested, but when it begins later it usually persists for years. In middle life the ozæna often grows less intense and toward fifty it may disappear. Very few cases can be claimed as cures.

Treatment may be summed up in one word—disinfection. The first step is to cleanse the passages thoroughly from all retained secretions. This may be done by douching, syringing, or spraying, though occasionally it may be necessary to detach hard crusts by mechanical means. The simplest instrument for this is a probe with cotton wool wound round it, which can be passed along the turbinated bodies with a gentle rotatory movement. It is only in very rare cases that this is desirable. Syringing or spraying will always succeed if properly performed and persevered with. An alkaline disinfectant lotion should be sprayed through the passages until all accumulated secretions have been washed away and the offensive smell is no longer perceptible. The patient must be instructed to maintain this gain by the regular use of the lotion with a coarse spray apparatus. When there is any difficulty or when these measures distress the patient—which is a very rare circumstance—the first complete cleansing may be prolonged over several sittings. In many cases an intelligent patient will accomplish it himself. After the parts have been completely cleansed they should be carefully explored by anterior and posterior rhinoscopy in order to discover any erosion, ulceration, or other complication. Any such lesions must be properly treated. In their absence the next point is to make the sprays more stimulating as well as disinfectant. The permanganates may be employed for this purpose; iodine may sometimes suit; either of these will perhaps exercise some influence on the membrane. Iodoform has been extolled, but its own odor is very offensive; it can be used by insuf-

flation or in bougies; Woakes employs a solution in ether as a spray. The same objection to the odor applies less forcibly to carbolic acid, but it is said to impair the sense of smell. For this, resorcin may be advantageously substituted in a one-half-per-cent. solution. Chloride of sodium has been largely employed especially as a douche, but I cannot recommend it. Chloride of ammonium is perhaps better, but this salt is more frequently applied in vapor, as Cohen recommends. Whatever spray is found to agree best must be perseveringly used two or three times a day so as to keep the nares free from odor. After a few months it may be found possible to do this by spraying only once a day. Instead of sprays, powders are recommended as stimulants. Lefferts and other American specialists recommend galenga and sanguinaria. They must be mixed with two or three or four times their weight of starch, gum acacia, or other indifferent powder and applied by the insufflator.

Gottstein introduced the plan of provoking increased secretion by a tampon of cotton wool. One nasal passage is to be plugged for two or three hours in the morning, the other in the afternoon. A considerable flow of secretion is induced by this foreign body, dried crusts are thereby detached and the whole membrane, so to say, washed from within. At the first application the tampon may be retained for a day. If the effect is not produced it has not been thoroughly applied; it should be in contact with the membrane so as to stimulate it to secrete. Instead of using the cotton dry, I would recommend moistening it with glycerin in order to add the effect of its stimulating action on mucous membrane.

The galvano-cautery has been employed by Fränkel, Shurley, Sajous, Mackenzie, and others, but a condition of atrophy seems scarcely to suggest destruction of tissue, and if only stimulation be intended there must be some danger of exceeding the intention. Delavan reports favorably of electric stimulation by applying the positive pole to the nape of the neck and the negative wrapped in cotton wool to the nasal membrane. A current of four to seven milliampères brings on a flow of secretion in from five to fifteen minutes.

Lastly, I may name the multiple puncture or even scarification which I have mentioned above as effectual in hypertrophic rhinitis. In atrophy the incisions should be as shallow as possible, and puncture is preferable in this form.

Rhinorrhœa.

A few cases have been recorded in which a constant or nearly constant watery discharge from the nose has been of such an amount as to be peculiarly distressing to the patient. In some of the cases

the flow seemed continuous, in others it diminished or even ceased for a short time at varying intervals; in some it was accompanied by great irritation, in others by none; in some it came on suddenly, in others gradually; in some the discharge was irritating, in others bland. Some of the cases seemed allied to hay fever, some were connected with polypus, some were attributed to traumatism, and some seemed neuroses.

Etiology.—The readiness of the pituitary membrane on slight provocation to pour out excessive secretion is exemplified in catarrh, to which some individuals exhibit a peculiar susceptibility.

In an ordinary cold the discharge is sometimes so excessive as to amount to an *acute rhinorrhœa*. Many persons measure the severity of a cold by the number of handkerchiefs they are obliged to use in a day and will talk of twenty or thirty as not uncommon. I once attended a gentleman between fifty and sixty years old who, on taking cold, found the discharge so abundant that he discarded handkerchiefs and sat all day over a spittoon into which there was a constant dripping. This seldom lasted more than twenty-four to thirty-six hours, after which the cold passed rapidly through the remaining stage. But it would scarcely be surprising for an attack of this kind to be followed by a chronic discharge, and some cases of rhinorrhœa have thus begun with a cold. Sir James Paget has recorded (*Medical Press and Circular*, 1878) a case in which the discharge, which had amounted to eight or ten ounces a day, decreased, and at length ceased altogether for a month when the patient died of an attack of meningitis. At the post-mortem examination a number of small round polypi were found in the left antrum (the side affected). It is not easy to see how these polypi could have caused the rhinorrhœa, which, moreover, had ceased a whole month before the fatal illness. In some cases the disease has evidently been of nervous origin, and possibly so in this, as the patient died of meningitis. A lesion of the brain or in the ganglia or along the course of the nerve is suggested by some cases, while in others the sympathetic system may be involved. Nearly all the patients have been adults and most of them neurotic. To the recorded cases I may add the outline of one.

In November, 1878, Mr. P. came to me with a request from his attendant that I would take him in hand and do what was possible to relieve his sad state. He was evidently of a highly neurotic disposition and said he was in despair and had contemplated suicide. He had for months been troubled with a constant discharge, day and night, from both nostrils in front and into the throat. He estimated the amount at two pints in the twenty-four hours, but so large a proportion flowed into the fauces that it was impossible to be accurate. At night it saturated the pillow. He could not lie on his back as it

produced spasmodic cough. The membrane looked rather pale and sodden, very slightly swollen. The fluid could be seen trickling into the pharynx. Nothing further was discovered by careful and repeated examinations. The discharge was quite bland and unirritating, never once excoriating the lip. Even the pharynx was not congested. The fluid had a specific gravity of 1.010 to 1.015, and contained a little albumin and mucin with traces of sodium chloride and phosphate. This gentleman came to me at intervals until the following February after which he ceased to attend. During these months there were fluctuations in the amount of discharge so that at times he was much encouraged and at others depressed. Some months afterwards he called to explain his absence which was caused by an attack of typhoid fever from which he recovered very slowly, but when convalescence set in he found to his great satisfaction that his nasal trouble had completely disappeared.

Treatment must entirely depend on the opinion formed of each individual case. If the cause can be ascertained, we have some indication, but as that is scarcely ever discovered we have none and clinical experience offers us no guide.

Chronic Purulent Rhinitis.

Störck has described a disease of this kind under the name chronic blennorrhœa. The disease begins with purulent secretion from the nose and extends through the pharynx to the larynx and trachea and even to the minute bronchial tubes. It is only on reaching the larynx that it becomes serious, leading to ulceration and sometimes to adhesions of the vocal cords. The disease seems confined to the peasantry of Poland, Galicia, Wallachia, and neighboring districts, and the patients all seem to have been poor and dirty. Tracheotomy has sometimes been necessary.

Epistaxis.

This word was applied by Hippocrates to bleeding drop by drop from any part and continued to be used in that sense down to the time of Vogel who proposed (1764) to limit it to hemorrhage from the nose. This restriction was gradually adopted and is now universal.

Bleeding from the nose is constantly met with to so slight an extent as to be looked upon as trivial, but it may be so profuse as to endanger life.

Etiology.—Epistaxis is common in the young usually from plethora, and in the old from vascular degeneration. The most frequent cause is injury. Blows, falls, picking the nose by children, passing bougies, the strain of violent exertion, coughing, retching, sneezing,

and blowing the nose may any of them bring on an attack of epistaxis. Slight deformity of the septum may give rise to local lesion, *e.g.*, erosion covered by incrustation, the removal of which by picking or otherwise may be followed by bleeding. Neoplasms and foreign bodies in the nose or in the naso-pharynx are also causes of hemorrhage. Constitutional defects are causes of grave import. A leading one is hæmophilia, epistaxis occurring in about half the cases. Leukæmia is perhaps the next most frequent state producing this symptom. Plethora has been alluded to, but anæmia must also be reckoned as a cause. Purpura, scurvy, phosphorus-poisoning, organic diseases of the heart, kidneys, or liver, may cause epistaxis, as may indeed any obstruction to the circulation sufficient to cause the vessels to give way where the resistance is least. Epistaxis often ushers in fevers or occurs in the course of them, and is regarded by many as critical. Probably the epidemic epistaxis mentioned by Morgagni was of this kind. Mental emotion sometimes precipitates an attack. Besides all this the hemorrhage may be vicarious.

Pathology.—The delicacy of the membrane and its free vascular supply explain the frequency with which the vessels give way. A varicose state of the vessels has been noticed in some patients known to be subject to epistaxis. Authorities differ as to the most frequent seat of the rupture—some finding it on the septum, some on the outer wall, others on the floor. It seems rare on the turbinated bodies.

Symptoms.—The attack is often preceded by a feeling of fulness, flushing, and throbbing in the head, and other prodromes recognized by the patient. Usually the blood drips from one nostril, but when it depends on blood changes, as in fevers, from both. Sometimes, with a free flow and some clotting, part of the blood may pass round the septum to the other side. A quarter to half an ounce is perhaps a common quantity, but much more may be lost, so that frequent attacks may even undermine the constitution. Sometimes dangerous syncope occurs.

Diagnosis.—Both nostrils and the naso-pharynx should be thoroughly explored so as to discover if possible the source of the hemorrhage. Patients are sometimes alarmed by coughing up a little blood in the morning which has trickled down the throat in sleep, and the rhinoscope may reveal that the bleeding took place in the nose or naso-pharynx where some streaks or clots still remain.

Prognosis.—The immediate risk from loss of blood must be estimated by the general symptoms, but in forming a judgment from the state of the pulse or the occurrence of syncope allowance must be made for the alarm of nervous patients with a first attack. The re-

mote prognosis will depend on the cause, whether local or general. In the latter case it resolves itself into the prognosis of the disease of which it is a symptom.

Treatment.—Slight hemorrhage in robust youths need not be interfered with. So, too, in difficulties of the circulation from cardiac or other disease. In both these cases the bleeding may be beneficial and its arrest may bring on unpleasant symptoms. It is well, therefore, not to interfere unless faintness seems impending. In vicarious hemorrhage every physician would be reluctant to interpose unless symptoms compelled. When interference becomes desirable, if the bleeding spot can be discovered, pressure with the finger will arrest it, or it may be touched with a stick of lunar caustic. In obstinate cases the actual cautery has been recommended, but pressure if continued will succeed. When such a spot cannot be found, pressure on the nostril so as to hold the ala firmly against the septum will often suffice. Change of posture may be of equal service. The patient often sits with his head bent over a basin, which is about the worst position he can adopt with comfort, as bending the neck tends to compress the jugulars and so impede the returning circulation. A semi-reclining posture on the affected side with the head inclining forward just enough to let the blood escape from the nostril is probably the best. The upright position has been recommended as lessening the flow of blood to the head, and holding the hand of the bleeding side above the head is said to diminish the force of the current. The chief advantage of these plans is probably that the patient can with one hand at the same time compress the nostril.

The *direct* application of cold to the nose has a very powerful depressant effect on the circulation, and the immediate effect in contracting bleeding vessels is well known. Ice if at hand may be applied or cold water may be snuffed up or used as a douche. So also the hemostatic effect of hot water may be utilized by fomentations, or water at 120° F. may be applied directly to the membrane by the douche or syringe. The *indirect* use of cold depends on reflex action and should therefore not be prolonged. This is the philosophy of the large key or other metallic substance slipped down the back which is still an article of popular faith. A lump of ice held for a few seconds at the nape of the neck would be more effectual. To prolong this use of cold is an error. Therefore Chapman's ice-bag must not be used, but on the contrary the warm water bag. Dr. Chapman's theory is that the vaso-motor circulation is controlled by heat or cold to the spine, the former stimulating the centre and so producing contraction of vessels at the periphery, the latter acting in the converse manner.

Astringent substances have been freely used either in powder or liquid. Of these alum has the advantage of being usually at hand. Tannin can be easily snuffed up and should be diluted with powdered gum so that it may adhere to the bleeding surface. The mixture of the powder and the blood, however, make an objectionable magma, and that formed with iron perchloride is still worse. Syringing and spraying with liquids are cleaner and probably more effectual, for in them the influence of cold is added to whatever may be that of the styptic. I therefore advise first a solution of alum, ten to twenty grains to the ounce. If thoroughly snuffed up it will often be so effectual that the bleeding ceases before a spray producer is procured. Antipyrin has been used in the same way—fifteen grains to an ounce. Pledgets of cotton wool are sometimes used to convey styptic powders or liquids, but they probably act, at any rate partially, by plugging or by pressure. The powerful influence of cocaine in contracting vessels may be utilized by a tampon or spray of a four-per-cent. solution.

When none of the foregoing measures succeeds we must plug the nares. To accomplish this easily an air-bag of rubber (rhinobyon, Fig. 16 *B*) which could be inflated after insertion has often been recommended. Several plugs of this kind have been contrived since Frank at the beginning of the century used a portion of a hog's intestine and distended it with water. Unfortunately these appliances when wanted are seldom at hand and in working order, skin and india rubber soon becoming defective when laid by. Iodoform gauze is preferable to lint. A long slip, about an inch broad, can be wound round a probe and passed through the nostril to the posterior orifice, when the probe is to be withdrawn and small pledgets of gauze pushed in until the cavity is full. To facilitate removal the separate pledgets may be attached at intervals to a string, making a kite-tailed tampon as suggested by Moure. Should hemorrhage continue we must proceed to plug posteriorly. For this Bellocq's sound (Fig. 16 *A*) may be used, but a female or Eustachian catheter will do as well. With this a thread attached to a stout ligature or string is passed along the floor of the nose into the pharynx where the end is seized and pulled through the mouth. A plug is then attached to the middle of the string and carried to the

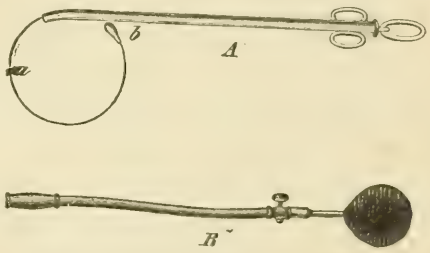

Fig. 16.—Instruments for Plugging the Nares. *A*, Bellocq's sound; *B*, rhinobyon or rhineurynter.

posterior orifice by traction on the nasal end. The buccal end is brought out and fixed so as to be ready when wanted to remove the plug. This should not be left more than forty-eight hours and must be removed with the utmost gentleness so as not to disturb the clot. A little tepid water or alkaline lotion passed gently through the other nostril will help to loosen the plugs, and a disinfectant astringent wash may be used for a day or two afterwards. Occasional unpleasant and even serious results have been recorded as caused by plugging, but they have probably been due to leaving the plug too long in place or to saturating it with styptics.

Constitutional treatment is called for in cases of repeated epistaxis. Medicines to contract vessels, such as ergot, may be tried. Turpentine is valued by many as a hemostatic; quinine is indicated when malarial influence is present, saline purgatives in plethora, and so on. When fatal syncope seemed impending transfusion has restored the patient, and Mosler records a case of hæmophilia in which transfusion arrested the epistaxis and removed the tendency to repeated hemorrhages.

New Growths.

These are frequent in the nasal passages and may be either benign or malignant. They have been noticed from the earliest times, and indeed they could hardly be overlooked, when of any size, on account of the obstruction caused to respiration, and the fact that they are frequently visible on the most cursory examination and sometimes protrude from the nostril. Hippocrates removed them by a piece of sponge fastened to a string passed into the pharynx and pulled back through the nostril.

Mucous, Vesicular, or Gelatinous Polypi.

These are the most common of all nasal growths. They are soft, pulpy, semi-transparent, pale or pinkish, yellowish or grayish. They may be single but are more often multiple, and frequently occur on both sides. They are usually pedunculated and easily moved, as they hang from the nasal wall by their narrow pedicles. They may be round, oval, or pyriform, sometimes are lobulated fitting the cavities. They may be quite small or may completely obstruct the passages. Their most frequent origin is the membrane covering the middle turbinated body or the immediate neighborhood; they are rarely attached to the septum, though sometimes they are pressed so closely on this division that they almost appear to be so. Now and then, too, these polypi get fixed to other points by pressure and fric-

tion, though this is more frequently the case with other growths. Zuckerkandl found in thirty-three cases examined post mortem only three growing from the septum, all the others being on or close to the middle turbinated bone.

The *cause* of the development of the growths is unknown and the numerous suggestions that have been made must be looked upon as guesses.

Symptoms.—At first stuffiness, increased secretion, and sneezing give the patient the idea that he always has a cold. As the polyp grows obstruction to the respiration becomes prominent, and this is often intermittent and apparently influenced by atmospheric changes. Hanging loosely in the passage polypi often produce the sensation of a foreign body moving about, and may even give rise to a flapping sound. Occasionally they seem to form a valve arresting the current of air in one or other direction. Important reflex symptoms may depend on the presence of polypi. Smell is impaired and often abolished. If left alone the tumors go on growing until they completely fill the nasal fossæ, and some are forced out of the nostril or back into the naso-pharynx, or they expand and force forward the alar cartilages and in some instances the nasal processes of the superior maxillary bone.

Pathology.—They belong to the myxomata. The investment is formed by the epithelium of the nasal membrane and therefore often ciliated. Within are glandular and connective-tissue elements, but there is considerable variation in these. Thus the glandular structure is in some polypi abundant, in others almost absent, and a delicate connective-tissue reticulum encloses a few myxomatous cells. The gelatinous intercellular substance which makes up the bulk of the growth contains at an early stage oval and round cells, which later on become fusiform or elongated and nucleated or more frequently granular.

Diagnosis is easy except in the earliest period, but even then careful rhinoscopic examination under cocaine may detect the growth. Cartilaginous and osseous growths are so hard as to be easily distinguished from these soft polypi. Fibromata, too, are harder, more resistant, redder, and often give rise to epistaxis. Malignant growths are distinguished by their color, fixity, and readiness to bleed on being touched; they cause a good deal of pain and bring about disfigurement, they grow rapidly and the discharge from them is offensive. Abscesses of the septum and hæmatomata have been reported as giving rise to mistakes but require little attention to differentiate them. A foreign body will give rise to inflammation and fetid discharge. Pathological curiosities such as distention of

the ethmoid cells and hernia of the dura mater and brain through the cribriform plate, discovered by Cruveilhier at an autopsy, are too rare to call for more than mere mention.

Prognosis is favorable, as the growths cause more annoyance than risk and can be removed. Recurrence is, however, very probable. Spontaneous expulsion has happened, but so rarely as in no way to affect the indication for removal.

Treatment.—From early times attempts have been made to dry up polypi or to destroy them by caustics. Even in modern times favorable reports have been published of such treatment as by insufflations of tannin (Bryant), silver nitrate (Nélaton), zinc chloride (Erichsen), iron chloride (Robinson). When operation is declined these agents may be tried, but it would be better to introduce them into the substance of the tumor. For liquids, the hypodermic syringe is sufficient. For chromic acid, a needle on the point of which the crystals have been fused can easily be introduced. This plan has been successful, but it is tedious and the resulting slough is offensive. Operation is therefore preferable, especially as the risks are inconsiderable. Removal by forceps is the oldest operation and, notwithstanding all that has been said against it, is well adapted to some cases. The ill consequences reported have been due to the use of unnecessary force and to a blind groping for the growth. With our modern appliances they should not occur. The patient may be seated as for laryngoscopy, with the tip of the nose somewhat raised. All possible information having been obtained under full illumination the forceps should be carried along the floor or septum just beyond the growth. The blades are then opened and turned upwards and outwards so as to catch the polypi as near the base as possible. A steady, rotary motion is then employed to twist the tumor off its attachment and withdraw it. Now and then a polyp may be partly withdrawn from the nostril without the attachment to the membrane giving way. Then the pedicle can be easily seized by another pair of fine forceps or may be divided with scissors or bistoury. The forceps should be slender but strong and may be toothed and arranged to lock. Stoker's rotatory forceps, Gant's vine scissors, and Mackenzie's punch forceps have good points, the last being also bent at a suitable angle to keep the operator's hand from intercepting his light—a point of importance in all nasal operations. The defenders of evulsion have gone so far as to say the removal of a lamina of bone which has sometimes occurred is an advantage and tends to prevent recurrence. This I cannot indorse. The growth does not originate in osseous tissue, and though the accident may be of little consequence, it is better it should not happen.

The écraseur has of late years largely displaced the forceps. It is easy of application, prevents hemorrhage, and does not injure the healthy tissues. A loop of wire is passed over the growth, carried as near as possible to the attachment and then drawn home. Jarvis' snare is a most satisfactory instrument. It has been said to be slow in action, but this is the best recommendation when hemorrhage is to be apprehended. The wire generally used is No. 5 steel piano wire, but other sizes may occasionally be employed. Dr. Jarvis has shown the great superiority of highly tempered steel wire such as is used for pianos over common unannealed wire.

The galvano-cautery may be employed in the form of a loop, but it is far more difficult to apply than the stiff cold snare and has few advantages, so that only those constantly using the galvano-cautery will care to resort to it. Some endeavor to destroy the growth by using a flat electrode, but it requires many sittings and the process is not agreeable. Moreover, I have seen very unpleasant consequences of applying the actual cautery in the nose.

Electrolysis has also been used, but it is even more tedious. The ancient plan of dragging a piece of sponge through the nares has been revived in this generation, but is not often resorted to, though it is simple and successful results are reported.

Fibromata.

These growths are dangerous from the hemorrhage they excite. Epistaxis is a constant symptom and the vascularity of the tumors is such that all interference with them gives rise to profuse bleeding. They of course cause stenosis and there is usually sanguineous and muco-purulent discharge. External deformity occurs as the growth advances—frog face or other deformity according to the position; exophthalmos has several times been observed. Happily fibromata are rather rare in the nose. Their pathology is the same as when occurring in other localities. The only treatment is extirpation, but this is not without risk. The galvano-cautery or the cold wire snare must be used. With the latter the closing of the loop should be done very slowly and interruptedly. Dr. Jarvis once took five hours over the snaring of a fibroma and was well rewarded by success with the loss of only a little blood.

Papillomata.

These growths are rare in the nares. Hopmann indeed reported 78 out of a total of 430 cases of benign growths and he states that Schäffer found 20 out of 182. Zuckerkandl found only 1 out of 39,

a proportion much nearer that met with by others. Perhaps some cases may have been confounded with mucous polypi. When at the junction of the mucous membrane and skin, they are harder than when in the interior. They resemble the same growths in other localities and may be destroyed by caustics or removed by the snare.

Enchondromata.

Enchondromata of the nose are very rare. They produce obstruction and deformity in the same way as fibromata, which they somewhat resemble. Their march is, however, slower and without epistaxis, but the discharge is sometimes offensive, probably from its exit being impeded. They give the hard, dense sensation of cartilage to the touch, are yellowish or pinkish in color, have a nodulated surface, and usually spring from the cartilaginous part of the septum. They may be extirpated by the snare and are not likely to recur.

Osteomata.

Osteomata sometimes start in the upper part of the nares or in the accessory sinuses. In their progress they soon cause deformity, sometimes even before obstruction is noticed. Epistaxis occurs in some cases, not in others. Fetid discharge as the result of retained secretion, ulceration, or necrosis is observed. Pain produced by pressure will naturally vary with the progress, and anæsthesia has been reported as caused in the same way. Sometimes these growths become separated from their attachment and are then movable. The diagnosis can sometimes be determined by the finger and probe, but a small piece must be detached and the microscope be called in to distinguish osteoma from osteosarcoma. Some present a cancellous structure but others are like ivory. The former can be crushed by forceps and removed piecemeal, but to extirpate the dense ivory growths it may be necessary to perform Rouge's or some other operation in order to gain access to them.

Exostoses.

These are to be distinguished from osteomata. They are much more common and do not often grow to a large enough size to cause serious inconvenience. If necessary they can be separated with forceps or attacked with the dental drill.

Adenomata.

These do not seem to occur in the nares in a pure form, though glandular tissue is found in adeno-sarcoma and other mixed neoplasms.

Cystomata.

Cystic tumors are exceedingly rare, but cases have been recorded by Watson, Lefferts, and Johnson.

Angiomata.

These, too, are very rare and some of the recorded cases were probably vascular tumors of a different formation. One of C. Wagner's seems to have been very mixed, part of it myxoma, another part myxo-fibroma, and a third part cavernous angioma. The diagnosis is not very difficult, especially when the growth is within reach of the finger. The tumors are apt to give way to a probe and bleed profusely. In removing them the one danger is hemorrhage and therefore the snare or galvano-cautery should be used.

Malignant Growths.

These neoplasms in the nares do not differ in their nature and course from the same growths in other parts of the body, except so far as they are influenced by the locality. Sarcoma is the more common form in the nose, carcinoma being much less frequent. They may originate from the septum, the floor, or the outer wall. Like other growths they produce obstruction with alteration of the voice and impairment of smell. There is usually a fetid discharge, epistaxis is frequent, and pain is often severe and located chiefly in the infra-orbital region. With increasing growth deformity comes on. The bridge of the nose is pushed forward and the bones are separated. Pressure on the inner wall of the orbit may produce exophthalmos. The cribriform plate has been destroyed and the growth has then penetrated the brain. If it extend backwards into the naso-pharyngeal space, characteristic symptoms will arise. Sarcomata grow very rapidly, are usually single and sessile, soft, smooth, dark colored, and very vascular, bleeding on the slightest touch. Carcinomata mostly begin as small warty growths or pimples, soft, friable, of a reddish color, with a tendency to ulcerate, the ulcers having raised, hard edges and a sanious base. Enlargement of neighboring lymphatic glands follows sometimes but not invariably. The diagnosis must be settled by submitting a portion of the growth to microscopic examination.

Treatment.—The only effectual plan is complete extirpation, if possible, but the outlook is gloomy, for recurrence will most likely take place. Moreover, it is often impossible to extirpate the growth.

A small sarcoma may perhaps be removed *per vias naturales* and the point of attachment can then be cauterized. But generally a preliminary operation to expose the tumor has to be undertaken, for partial removal only provokes increased and rapid growth. In carcinoma surgical interference is more likely to shorten than to lengthen life. It may be added that we are often not consulted until the only measures available are mere palliatives, the advanced state of the disease and the condition of the patient forbidding interference.

Affections of the Septum.

The septum has of late years attracted a degree of attention which seems in some respects disproportionate to its size and importance in the economy. It has been seen to participate in various affections previously described, but some other lesions may now be noticed to complete its pathology.

Abscess.

This may be acute or chronic and usually affects both sides, so that we have to deal with two red and tender swellings which are generally situated at the anterior part and are symmetrical. They are almost always due to traumatism, so that inflammation of other parts is at first present. Acute cases set in within a few hours of the injury and may cause some pyrexia. Obstruction of the breathing and altered voice with pain and tenderness are the chief symptoms, to which may be added conjunctivitis attended with profuse lacrymation. Chronic abscesses begin a week or two after the injury and pursue a slower course. The swellings are lighter in color, less painful, not very tender, and seldom give rise to febrile excitement.

The *treatment* of these abscesses is that of the same condition elsewhere—evacuation and drainage, which are easily accomplished. On opening one a probe will almost always show that perforation has occurred. The other should then be evacuated and a thread or a twist of cotton wool passed through the communicating opening. The healing is generally rapid, but the perforation, which is of no consequence, will remain.

Hæmatomata.

These may be caused by traumatism; they may present the symptoms and appearance of abscesses, but are of a darker color. A blood tumor is usually a complication of fracture of the septum. The blood is effused between the membrane and the cartilage be-

neath, forming bluish or purple, smooth, soft tumors in which fluctuation from one side to the other may be detected. When large they may protrude from the nostrils. Usually they soon degenerate into abscesses. Luc and Péan have each reported a case of unilateral hæmatoma which appeared to come on without injury.

If *treated* by cold at the earliest moment small hæmatomata may become absorbed. If this does not take place in the first forty-eight hours, one of the swellings may be punctured at the most dependent part, and if pressure with the finger does not suffice to empty both, the other should also be opened. Perforation, as in the case of abscess, is almost certain to be left.

Perforation.

We have seen that perforation of the septum may be produced by abscess or hæmatoma; it may also result from tubercle and other lesions. Most frequently it originates in syphilis—so frequently in fact that at one time it was regarded by many as a characteristic of that disease. How erroneous that idea is needs no further demonstration. Perforation through the bony portion of the septum is generally the result of syphilis, but it may succeed injury and has been known to follow idiopathic periostitis. In addition to the causes already named, perforation of the cartilaginous portion may be due to local inflammation, ulceration, or even simple erosion. Sometimes a local irritation provokes a picking of the nose and the membrane is injured; a thin crust or scale is in its turn picked off and eventually the cartilage is reached. A slight projection of a part of the septum into one nostril has been thought sufficient to set up irritation or even erosion. The current of inspired air impinges on the projecting surface and the dust becomes deposited in the same place and so irritation may possibly be initiated; but this explanation is not entirely satisfactory, for it does not show why these small projections should set up irritation after existing for many years without having done so. Cases of congenital perforation, suggestive of arrested development, have been recorded. Occasionally perforation has been met with in a septum that was so far out of place that the air current passed through it and produced a whistling noise. Otherwise these communications between the nostrils are of no importance and are merely the evidences that some lesion has at one time existed. During their formation, indeed, slight hemorrhage may occur but may scarcely attract notice. The edges heal quickly and the only result is a hole with smooth, rounded borders which produces no inconvenience and of which the patient is generally unconscious.

Deviations.

It is to certain abnormalities in shape or direction that the septum owes the place to which it has of late been raised. Deviation from the median line, or distortion due to violence, to unequal development, or to any other cause, in short any conformation which differs in any degree from the assumed ideal has been blamed as a cause of various diseases and subjected to surgical procedures.

The assumed ideal is a septum dividing the nostrils into perfectly symmetrical cavities. Now considering the many cases in which the symmetry of corresponding parts of the body is more or less imperfect, it may be interesting to inquire into the frequency of deviations of the septum. Theile found symmetry in only 29 out of 117 skulls he examined—*i.e.*, in 73.5 per cent. there was deviation. Semeleder found in 49 skulls asymmetry in 79.5 per cent., and Harrison Allen 68.9 per cent. of 58 crania examined. Zuckerkandl examined 370 skulls and found in 140 deviation—37.8 per cent. Mackenzie examined 2,152 crania in the museum of the Royal College of Surgeons, and of these 1,657 presented want of symmetry—76.9 per cent. Loewenberg reckons that symmetry occurs only in 1 skull out of 7. These statistics relate only to the bony septum, but during life it is naturally enough assumed that the cartilaginous portion often deviates when the bone remains straight. In a discussion held during the sixtieth meeting of the Society of German Naturalists and Physicians, Dr. Heymann stated that deformity occurs in ninety-nine per cent. of the persons examined. Do not these figures suggest that nature is not so much enamored of symmetry and that noses may be normal although the two passages are not exactly alike? Zuckerkandl observed that the barbarous or semi-barbarous races are less subject to such deviations than the more civilized, and this curious observation was confirmed by Mackenzie and Harrison Allen. Zuckerkandl says that the deviation is not found in children under seven years of age, but others assert that some cases are congenital.

It is not intended to imply that deformity of the septum is always unimportant, for we know that the condition is sometimes extreme and gives rise to serious symptoms. Thus the whole nose may be twisted so far to one side that the disfigurement is noticed by every one. In other cases there is very slight or no external effect. Inspection anteriorly of course reveals the asymmetry and it may be reflected in the rhinoscope, although the defect never extends to the posterior part of the septum. A projection into one nostril of course implies an opposite state of the other. Cases in which there is a

prominence on one side without a corresponding depression on the other are generally due to the formation of a ridge along one of the sutures, and most frequently at the junction of the vomer with the superior maxilla or with the cartilage of the septum. These outgrowths, sometimes called *exostoses*, may be bilateral. Thus Mackenzie found ridges of this kind in 673 of the skulls he examined. In 375 the projection was on the left side; in 231 on the right; in 67 on both sides.

Whatever the nature of the deformity, when there is encroachment on one nasal passage obstruction will be produced in proportion to the encroachment. In some cases the nostril is completely occluded. The nasal respiration is impeded, the secretion is hindered from escaping, the voice assumes the nasal twang, the turbinated bodies often atrophy, and post-nasal catarrh and atrophic rhinitis may follow. On the other hand hypertrophy of the turbinated bodies may be produced, generally on the more open side, so that when there is a large bulging on one side, almost or quite occluding it, the tissues over the turbinated bone on the concave side enlarge and partially or entirely fill the cavity.

Diagnosis is at once determined by inspection and comparison of the two sides. A probe may also be used after the application of cocaine. Seiler devised a septometer to determine the thickness of the septum at various points. Strong illumination of one nostril enables us to note through the other the degree of translucency. Polypus is distinguished by its softness, mobility, color, and elasticity. Other tumors are more or less movable and have their own characters, while septal deflections are rigid, immovable, and in no degree separable.

Treatment.—The indication is to remove obstruction. In slight cases of deflection of the cartilaginous septum in young persons, gentle pressure with the finger repeated frequently has been advised. When the object is not to remove deformity but to prevent it being a source of disease, an artificial perforation has been established with success. Punches have been used for this purpose. When the bony septum is the seat of the deviation and there is deformity of the face, Adams' operation is suggested; but it is no easy matter to refracture at the point of the old injury, and the necessary wearing of splints, plugs, or clamps is not a pleasant prospect. Forcible replacement of a distorted septum is often a simple procedure, but maintaining the part in the proper position will tax the ingenuity of the surgeon and the patience of the sufferer. The more acceptable plan seems to be to excise the projection and so free the passage. For this purpose knives, scissors, snares, forceps, gouges, saws, trephines, and other

instruments have been devised, and success is reported with all, showing that the end may be accomplished in various ways. Even the dental engine has been recommended, though that would seem more appropriate for dense, hard exostoses. Bosworth advocates a fine saw which has the merit of simplicity and with which he reports numerous successful operations. Having thoroughly anæsthetized the part he proceeds to saw off the projection "as we saw a board out of a log." The bleeding is profuse for two or three minutes but is arrested by the clot, which he directs the patient not to disturb for three hours. In only two cases has he had to plug. Objections have been offered to this plan that it may result in ulceration or hard cicatrices, but he says he has never seen either. The mucous membrane reforms over the cut surface, and if this be smooth and level the cure is complete.

Dislocation of the Columnar Cartilage.

Under this term two cases are recorded by Bosworth in which a small oblong plate of cartilage lying parallel with the lower border of the septum and apparently supporting it, became displaced and partly closed one nostril. He dissected out the plate, removed some redundant mucous membrane, and closed the wound with fine sutures.

Synechiæ.

These are bands of tissue connecting parts which are normally separate—such bridges, both membranous and osseous, have connected the septum with the floor or one of the turbinated bodies, and are therefore mentioned here, though they may span other parts of the passage as described by Zuckerkandl. In the two thousand one hundred and fifty-two skulls examined by Mackenzie at the Royal College of Surgeons were found four cases of bony synechiæ. The condition is therefore a pathological curiosity. It could easily be remedied by dividing both attachments of osseous synechiæ with nasal bone-forceps while the membranous variety would only require a knife.

Neuroses.

The sense of smell may be either impaired or altogether lost—anosmia. On the other hand it may be rendered preternaturally acute, a condition to which the term hyperosmia has sometimes been applied. Again the sense may be altered or perverted—parosmia. In this section we must also consider affections of the fifth nerve, and the nasal reflexes which have of late attracted so much attention.

Anosmia.

This term, signifying absence of smell, is applied to the loss of that function and is also extended so as to embrace cases of impairment or partial loss. The affection may supervene from disease or injury to the bulb or to any part of the nervous tract. It may also occur from any circumstance which prevents odoriferous particles approaching or acting on the terminal filaments—obstructive as distinguished from essential or nervous anosmia. Interference with the function of the fifth and seventh nerves may impair the function, as may be observed in the dry stage of catarrh. A moist state of the nasal membrane is essential to the perception of smell.

Etiology.—Anosmia may be caused by prolonged exposure to powerful odors; the over-stimulation seems to exhaust the nerve, and such cases are perhaps analogous to snow-blindness. It has been thought that the effect of ammonia was due to direct action on the endings of the nerves, but other powerful odors produce a similar result. Snuff-takers have sometimes been observed to lose partially or completely the sense of smell, but considering how widespread this habit once was, we have very little evidence of this special injury. The free use of the nasal douche has occasionally been followed by anosmia, probably from the liquids used being too concentrated or unsuitable. Alum is said to be particularly prejudicial. Stricker reports a case caused by ether. Sometimes this symptom—for anosmia is rather to be regarded as a symptom than a disease—has been associated with neuralgia of a periodic character and cured by quinine. The most common cause of temporary impairment of smell is a cold, and probably prolonged rhinitis is the most frequent cause of permanent loss of the function. Perhaps Schultze's cell processes are destroyed by the pressure of inflammatory products, or their function is so long interrupted that atrophy occurs. Althaus suggested that there might be hemorrhage into the nerve itself. Long-continued paralysis of the fifth nerve is likely to impair or destroy olfaction. Falls on the head have been observed to be followed by anosmia, and the olfactory bulbs have been found on post-mortem examination to have become severed from the brain. This lesion may be compared with congenital absence of the bulbs, of which examples have been recorded—the association of some with cleft palate suggesting an explanation of their mode of origin.

Besides these causes on the side of the nervous system there is the question of pigment. According to the researches of Dr. William Ogle, diminution of pigment probably leads to impairment of smell,

and dark animals have the sense in greater perfection than light ones. Very interesting facts have been accumulated pointing in this direction; but, on the contrary, albinos are not usually anosmic, though a case has been reported in which such a person lost the sense of smell in advanced age.

There remain the obstructive cases. Polypi or other growths or anything occluding the nostrils may interrupt the function, so may the falling in of the nostrils from paralysis. The odoriferous particles are thus prevented from impinging on the mucous membrane. These cases are seldom really complete. If the nostrils be opened mechanically and the odorous substance brought near, the patient may perceive the smell if able to sniff even a feeble current of air. As the membrane must be moist, the influence of the seventh nerve will be understood.

The *symptom* is a subjective one and therefore only learned from the patient. Olfaction is so closely associated with taste that loss of the latter is usually first perceived. Taste perceives sweetness, acidity, saltness, bitterness, but flavor is due to the coöperation of smell. Curiously enough, when smell is impaired the perception of irritants sometimes becomes more acute.

The *pathology* of anosmia has been partly anticipated above. Abscesses or tumors involving the olfactory bulbs, pressure upon them, their separation from the brain, or their complete absence readily account for essential anosmia. Another defect has been noticed—absence of the nerve branches supplying the pituitary membrane. Degeneration of the nerve trunk similar to the atrophic condition sometimes found in old age has been seen by Prévost.

The *diagnosis* is simple. The obstructive cases may be distinguished by discovery of the cause through the speculum and mirror. It is desirable, too, to note whether the defect is unilateral or bilateral, and to decide this, one nostril at a time must be accurately closed while to the other are presented substances with odors familiar to the patient. In nervous cases some other neurosis is usually present. Injury to the fifth or seventh nerve is almost invariably unilateral.

Prognosis in the obstructive form is favorable if the sense is not absolutely lost and the obstruction is removable. Anosmia from slight injuries to the head is said to offer a worse prognosis than from fracture of the base, probably because the concussion may have caused separation of the bulbs. Again, anosmia resulting from prolonged rhinitis offers less probability of recovery than the obstructive form. Thus the removal of a polypus has restored the sense after fifteen years of obstruction, but I know of no case of recovery from catarrhal anosmia of two years' duration.

Treatment.—In the obstructive form the treatment resolves itself into removal of the obstruction. In essential anosmia galvanism has been recommended but offers little prospect of benefit. The normal olfactory nerve is not stimulated with a current which is not powerful enough to cause pain, flashes of light, hissing noises, faintness, and giddiness, and such a remedy would be worse than the disease. What then could be expected from mild currents? Another recommendation is for the patient to endeavor to excite the nerve to action by the frequent presentation of different powerful scents. This the patient may be glad to carry out, and weak galvanic currents might also be tried, if desired.

Tonics should be given if indicated—one of the best is strychnine. This may be given as sometimes recommended in insufflations, but it must be remembered that the remedy is absorbed and produces the constitutional effects while it has probably no special local influence.

Parosmia.

A subjective sensation of a strange or disagreeable smell is often complained of by lunatics, and the question naturally arises whether in a given case it is a pure hallucination or may be due to some lesion affecting the olfactory bulbs or nerves. Such a sensation sometimes forms part of an epileptic aura. Lesions involving the bulbs may certainly cause parosmia. The symptom has been noticed in a case of cancer which destroyed the left olfactory tract. In another case a tumor had involved the bulb. In other cases of parosmia the cause cannot be traced, but it is evidently of a different kind, as the symptom may disappear. Sometimes the symptom occurs in persons in whom no other deviation from the normal state is noticed. The odor may be distressingly disagreeable or merely indifferent or even agreeable if it were only perceived for a few minutes at a time, but the most pleasant scents become offensive if persistently perceived. Now and then otherwise normal individuals find certain substances give them the odor of others. Mignonette and violets, for instance, have both been perceived as garlic, and other equally curious anomalies have been observed. One might suppose such cases to be somewhat analogous to those of color-blindness. Inflammatory changes in the nerve itself have been credited with the perversion, but we have no definite knowledge of such changes.

Affections of the Fifth Nerve.

The nasal mucous membrane may lose its sensibility in consequence of disease or injury of the fifth nerve, so that ammonia, for

instance, does not produce sneezing, which is ordinarily set up by any irritant. Thus sneezing is a common symptom in the early stage of catarrh; the flux which follows is, however, no doubt due to a vasomotor paralysis.

Treatment.—Mild galvanism may be tried, but the conditions are so uncertain that the only rational indication is to remedy any associated state of ill health.

Reflex Effects of Nasal Diseases.

Isolated instances of remote affections caused by nasal lesions have been recorded from time to time—notably a case by Voltolini. But in 1882–83 the late Dr. Hack contributed a series of cases to the *Wiener Medizinische Wochenschrift* afterwards embodied in his remarkably able monograph, and since that time rhinologists have pursued this subject with an ardor which has excited the smiles and sometimes the sneers of general physicians. Among the maladies which Hack traced to nasal reflex are redness and swelling of the integument of the nose whether temporary or permanent, circumscribed and transitory œdema, ciliary and trigeminal neuralgias, cephalalgia, migraine, photophobia, scotoma, muscæ volitantes, salivation, and even exophthalmic goitre. When sneezing and excessive nasal secretion with stenosis, alternating between the two sides, were associated with such troubles he looked confidently for disease of the turbinated bodies as the cause. Hypertrophy and hyperæmia of the nasal mucous membrane were the most frequent conditions he found—the latter more frequently than the former, perhaps as being due to vasomotor paresis. A case of aphonia is reported by Max Schaeffer, of chorea by Elsberg, and another by Jacobi, of epilepsy and asthma by several observers, and the list of reflexes is constantly receiving additions.

The explanations which have been suggested of these reflexes are not very satisfactory. Hack's is that various influences within or without the nose produce reflexly hyperæmic turgescence of the inferior turbinated bodies, in which condition on any slight irritation they excite a secondary reflex in remote parts. Moldenhauer objects to this that in cases of polypus or polypoid thickening there may be no hyperæmia. But others allege that these conditions favor reflexes while atrophic rhinitis does not. Dr. John Mackenzie finds certain "sensitive areas" which are "limited to the mucous membrane covering the middle and inferior turbinated bodies and the posterior half of the septum." In complete atrophy of the turbinated bodies reflexes cannot be induced artificially, yet cases of fa-

cial neuralgia and vertigo in atrophic rhinitis have been cured by nasal treatment (B. Fränkel, Schmaltz, Finne). Dr. E. J. Moure considers that reflexes arise from mechanical irritation; both Schiff's and Bert's experiments show that mechanical and also chemical and thermic irritation of the nasal membrane may reflexly affect the respiration, and Brown-Séquard taught that plunging the nose into cold water might arrest cardiac action and respiration. Some writers have evidently been conscious of the insufficiency of these suggested explanations, for they have called in general constitutional states to eke them out. Nor is this altogether unreasonable, for hysteria, the neurotic disposition, and other general conditions may well play their part in reflex affections. But while admitting this the general physician very properly observes that a number of the maladies said to be cured by intranasal treatment are amenable to other measures, and they are certainly inadequate supports for the extensive inferences that have occasionally been drawn from them. Aphonia and chorea, for example, are scarcely the diseases on which to frame an hypothesis. Vertigo is a symptom of such varying import that it is difficult to accept the explanation offered to the French Laryngological Society (1877) that it is due to irritation of the terminal fibres of the trigeminus. By laying down the broad assertion that any irritative or vasomotor disturbance in the nose is likely to cause reflex disturbance in any other part of the respiratory apparatus, we may explain spasm of the glottis, asthma, hay fever, etc. A very slight extension of the principle will cover facial spasm, headache, and all the neuralgias. Add to this pressure on peripheral nerves, whether by thickening or by new growths, and we need not wonder at the number of conditions to which have been assigned a reflex origin.

But if we take a more serious neurosis—epilepsy—the neurologist will not be disposed to deny that the fits may have ceased after the rhinologist's suggestion has been carried out; but that this is merely the direct effect of the cause being removed he will probably hesitate to admit. It seems to be a fact that some cases have permanently recovered, but in others the fits have returned after a more or less prolonged interval.

The *diagnosis* of nasal reflexes was determined by Hack by means of cocaine. Reflex phenomena produced by the probe before anæsthetizing ceased when the membrane was completely anæsthetic. Others make their diagnosis in the more difficult cases by the effect of treatment.

This *treatment* is of course local and frequently operative. It may therefore well be asked whether all the benefits said to fol-

low should be ascribed to the treatment of a local lesion or whether counter-irritation may not be the remedial agent. This point has been treated by Dr. McBride (*British Medical Journal*, January 29th, 1887). The mental influence of an operation on a neurotic patient may also be considered.

Foreign Bodies.

Children often amuse themselves by pushing up their nostrils beads, peas, small pebbles, or any other articles and occasionally such substances become impacted in the passage. Lunatics also seem to have a propensity to this habit. Foreign matters have also occasionally been driven into the nares from below by the act of vomiting or by an effort to prevent food "going the wrong way." Portions of knives, bayonets, and other weapons, bits of pencil, splinters of wood, etc., have penetrated by wounds from the outside and lodged in the passages. The variety of substances which have entered in one way or other is really remarkable.

Symptoms vary with the nature of the accident and the shape and size of the intruder. A small, hard, round, smooth body like a bead may remain a long time without giving trouble. A soft substance of similar size—say a pea—will swell up as it imbibes moisture. A haricot bean is reported to have germinated and shot out several roots, being mistaken for a polyp (Boyer). Pointed or irregularly shaped bodies provoke rhinitis. Headache, pain in the nose as well as pain in the cheek simulating facial neuralgia may be set up. Later on the rhinitis becomes chronic and fetid discharge ensues.

Diagnosis.—When a clear history is to be had there is no difficulty; but often this is not the case, for the accident may have been forgotten or may be concealed. It is therefore a golden rule in every case of fetid discharge to remember the possibility of a foreign body being retained, especially in young or insane patients. A thorough examination by anterior and posterior rhinoscopy should be made after the passages have been well sprayed out to remove mucus which is likely to be accumulated on the impacted body. A probe may be required, before using which local anæsthesia should be induced.

Treatment.—Blowing the nose and taking a pinch of snuff are popular notions which will probably have been brought into play before medical advice has been sought. An emetic, too, may have been administered, as it has sometimes been successful when the mouth was firmly closed during vomiting so as to force the evacuated fluid through the nares. Of course these methods are only resorted to when the accident is known and recent. When there is no information as to the entrance of a foreign body, no treatment has been

employed. Whether or no, when advice is sought and diagnosis decided the foreign body should be removed as soon as practicable. There is, however, no danger in some further delay if for any reason it should be desirable to defer making the attempt. Before the operation the passages should be thoroughly cleansed and then anæsthetized. In the case of young children, lunatics, and some other patients it may be desirable to give a general anæsthetic. The position of the foreign body having been precisely determined it may probably be grasped by a delicate pair of forceps. Should this not be practicable, a loop of the snare may perhaps be passed over it. If too large to be extracted whole it may be crushed with forceps or divided by the snare. Gross' hooks and spuds are useful for peas and soft bodies, and the screw may often assist. Some have succeeded by pushing the object backwards into the pharynx, but this plan is to be deprecated on account of the danger of its slipping into the larynx. With an intelligent patient the douche will sometimes dislodge the body, but as there is some risk of injury care should be taken not to raise the reservoir too high. Irrigation from behind by means of a suitable irrigator is preferable. So, too, a sound passed behind, guided by the rhinoscope, may dislodge and push the foreign body forwards.

Rhinoliths.

When a small foreign body becomes lodged in the nasal passages the salts of the secretion are apt to collect on its surface and so form a concretion which continues to increase in size just as calculi are formed elsewhere. Sometimes the centre contains only an albuminous liquid, and it is doubtful whether the nucleus is a softened foreign substance or the remains of a pathological product. There seems no reason why a particle of inspissated secretion may not become the nucleus of a concretion.

The *symptoms* are those of other foreign bodies but come on very gradually, and after a time they become more urgent as the calculus continues to grow and sometimes to attain a great size. Their shape is to a large extent determined by their position and therefore very variable. An offensive discharge is set up and obstruction gradually increases. In some cases they have attained such dimensions as not only to close the passage but to cause great distress by their increasing pressure culminating in external distortion. They consist of mucus and phosphate of lime, are usually softer interiorly and harder on the surface, but some have been found very hard throughout.

The *diagnosis* is not always easy. From osteoma rhinoliths may generally be distinguished by being somewhat movable, though some-

times they are very firmly impacted; also by less hardness so that a needle or probe will generally penetrate them to some extent. Their slow progress without deterioration of the health and without great pain distinguishes them from cancer, though mistakes have sometimes been made.

The *treatment* is that of foreign bodies—removal either whole or piecemeal.

Living Foreign Bodies.

Insects sometimes find their way into the nares and give rise to serious trouble. Flies may enter for a few seconds and deposit their eggs which are soon hatched. In tropical countries this accident is more frequent and is attended with great suffering and sometimes death. The destruction of the soft tissues is rapid and attended with excruciating pain which is continuous, preventing sleep. Caries of the osseous structures, convulsions, coma, and death succeed. In India the term "peenash" is applied to cases of this kind, but also to others in which there is foul discharge, so that the word is as indefinitely employed as ozæna has often been. The Lucilia hominivora is the most frequent intruder, but other muscidæ may also enter the nares with similar results.

Other living creatures have been known to gain access to the nares such as a leech, a centipede, an earwig. Ascarides may crawl up from the alimentary canal after death. But a few cases are recorded in which worms have been expelled from the nose during life. Probably they may have crept up all the way or possibly have been carried up from the stomach in the act of vomiting.

Symptoms.—Cases have been recorded, the nature of which was not suspected, until after much suffering, a number of larvæ were expelled from the nostrils. Probably the fly had entered and deposited its eggs when the patient was sleeping. Otherwise such an intrusion in tropical countries would naturally excite suspicion. The eggs are hatched in about twenty-four hours and irritation of the mucous membrane, sneezing, tickling, and formication are complained of. Then follows a sanious or bloody discharge with great pain, œdema of the face and eyelids, and swelling of the palate. The pain in the nose and frontal region is of an intense throbbing kind and has been compared by sufferers to a continual hammering. It seldom abates or ceases for even short intervals. Epistaxis is frequent. Destruction of the soft tissues is followed by that of the cartilages and bones, and meningitis may ensue. In more favorable cases, abscesses may form on the nose, and when these break or are opened the maggots escape, but before this some will probably have

been discharged through the nostrils. The number of maggots discharged is accounted for by the enormous number of eggs produced by the fly.

The *diagnosis* of myasis rests upon the discharge of the maggots.

The most effectual *treatment* is to wash out the passages with chloroform. This is of course painful, and inhalation of the anæsthetic may precede its use in the syringe. The chloroform may be diluted one-half, but if that fail may be used pure. Since Dauzat, an apothecary's assistant in Mexico, discovered the value of chloroform, the remedies formerly tried, such as turpentine and tobacco, have been quite superseded. After the chloroform, disinfectant lotions should be freely used to cleanse the passages. Morphine may be injected subcutaneously to relieve pain. Should the maggots reach the accessory sinuses it becomes more difficult to dislodge them and it may be necessary to consider the question of opening the antrum or trephining the frontal sinus.

Nasal Lesions in General Diseases.

Nasal symptoms occurring in the course of constitutional diseases present the greatest differences of intensity and significance. They may be so mild and of such brief duration as scarcely to attract the patient's attention, or, though somewhat troublesome, they may be of little import and be speedily cast into the shade by the graver general disturbance. On the other hand the nasal lesions may be so severe and fraught with such serious consequences as to rank as diseases rather than complications or manifestations of constitutional affections.

Nasal Syphilis.

A few cases of primary sore in the nostrils have been recorded, the patient's own finger having generally been the medium of infection. Secondary affections in the nasal passages are decidedly rare, but as they do not cause much inconvenience they may sometimes be overlooked. Tertiary lesions are more frequently met with, and the great and permanent deformity caused by their ravages may produce the impression that they are more common than statistics seem to indicate.

During the secondary stage no doubt hyperæmia of the mucous membrane often produces coryza, which is neglected as a mere cold, but sometimes lingers so long that the patient seeks advice. In some few cases mucous patches form and the secretion they provoke is exceedingly intractable and mostly of a muco-purulent character.

They do not affect the junction of the skin and mucous membrane here so much as at other outlets and should therefore be looked for in less conspicuous positions. They are, however, so seldom seen that some authorities seem sceptical as to their existence. Yet there is no reason to regard this membrane as exempt, and when patches are found in no way distinguishable from those in other parts, and particularly if coincident with characteristic skin affections, the diagnosis can scarcely be doubted. Superficial ulcers are perhaps more frequently seen, as they give rise to more irritation. They have been regarded as caused by the breaking down of mucous patches, but probably some of them may be due to superficial gummatous infiltration. This would be the more likely when they occur rather late in the secondary or as some would say early in the tertiary stage.

The later manifestations of syphilis in the nose are of far more importance, not only in themselves but on account of the deformity and other grave consequences which they produce. A gumma may appear on any portion of the membrane. On the septum it may attain such a size as to block the passage and present an appearance which might easily be mistaken for some other tumor. In this situation it is not painful, though some uneasiness may be felt as well as the sense of obstruction. On the turbinated bones it gives rise to considerable pain with nocturnal exacerbations. Gummata in the nose often seem slower in their march to ulceration than in some other localities, perhaps because they are not subjected to external irritation. Their early recognition is therefore doubly important as affording the chance of averting their consequences by treatment. The breaking down of the gumma establishes the destructive ulceration which is the grave feature of this stage of nasal syphilis. The pain is now much less and may disappear. There ensues a foul, profuse, sanguino-purulent discharge, mixed with crusts and shreds of necrotic tissue. Often large flakes of a white cheesy substance appear, especially after the ulcer has been cleansed; it has probably been retained in the upper passages. Sometimes spiculæ of bone or larger sequestra are separated and pass out of the nostrils or into the pharynx. If only the turbinated bones are destroyed, no deformity is produced, but when the vomer is extensively involved in the morbid process, the bridge of the nose may fall in, giving rise to an appearance suggestive of the feature having been crushed. When the cartilaginous septum is attacked rapid destruction may occur and another characteristic deformity be produced by the want of support of the tip of the nose which hangs loosely from the bony part above. Occasionally the columna is also destroyed, the two

nostrils being thrown into one, over which the tip flaps. This is rather exceptional, but I have seen still worse deformity, the whole of the nose having disappeared in a long neglected case and two small holes amid a mass of cicatrices serving as the nostrils. Cases are also recorded in which the disease has involved the superior maxilla, and the basilar process has been invaded, the cranial cavity laid open, and fatal inflammation of the meninges and brain produced. Occasionally perforation of the hard palate occurs, but whether this is a result of a downward extension of the disease or of a separate gummy deposit, seems uncertain. It might be anticipated that septic infection would not seldom follow the exposure of bone in the presence of continual purulent discharge. This result is, however, exceedingly rare, but Hermann Weber has recorded a case in the Medico-Chirurgical Transactions (Vol. XLIII.), in which post-mortem examination proved that it had occurred.

Diagnosis of tertiary syphilis of the nose is not difficult. Lupus when it begins on the mucous membrane may somewhat resemble it; but lupus usually occurs at an earlier age, the hereditary form of syphilis excepted, besides which the papules and tubercles of lupus are distinctive. The ozæna of atrophic rhinitis differs from the stench of syphilitic caries and can be removed by syringing, which, however thorough, will not get rid of the odor of the diseased bone. Gottstein's plugs remove the smell of ozæna but aggravate that of syphilitic necrosis. The history and the presence of other lesions or scars will of course assist.

Pathology.—The characteristic process seems to be infiltration of the hyperæmic and perhaps hypertrophied mucous membrane with small round cells. These cells extend through the whole depth of the membrane, pressing on the vessels and follicles so as to obliterate their channels and cause a dilatation beyond. The obliteration of the vessels is expedited by further infiltration within. Occasionally among the round cells a few of a spindle shape have been found. This cellular infiltration extends into other tissues sufficiently to obliterate the normal boundaries. The bones and cartilages near the diseased membrane become involved, the result being exfoliation from necrosis, or rarefying osteitis, or a plastic form of the latter. Sometimes, however, as shown by Sänger, the bones or cartilages may be attacked before the overlying membrane, so that we may meet with primary syphilitic caries in the nose analogous to that seen in the tibia. Moreover, E. Fränkel found in some cases post mortem molecular bony necrosis when the ulcerations were so small as not to be detected during life.

Prognosis is favorable in the early stages and even in mild terti-

aries, provided the patient will carry out a course of antisyphilitic treatment. When active necrosis is present, the case is grave, not only from the point of view of deformity but because the health has too often been greatly undermined. When this is not the case, the prognosis is that of tertiary syphilis elsewhere, except so far as regards the danger of deformity.

Treatment.—The manifestations of syphilis in the nose demand the same management as in other parts, the modifications of local measures being merely those necessitated by conformation. Coryza in the adult will usually subside under antiseptic washes or sprays. Mucous patches require the application of caustic. Solid nitrate of silver may be applied every day or two; tincture of iodine is preferred by some, but chromic acid is more effectual.

No local application should be made to a gumma which is to be treated only by constitutional measures. If accompanied by discharge, an antiseptic spray should be used.

Ulcers, even if only superficial, should be cleansed and iodol applied or a detergent disinfectant wash should be regularly used. Deep ulceration should be treated on the same lines but with more activity. The first point is to remove all crusts and discharge. This may usually be accomplished by alkaline carbolic sprays or irrigation, but at times a pledget of cotton wool may be used to expedite matters. Should the cleansing process have been painful or unusually difficult, nothing further need be done for one or two days except frequent spraying with carbolic or permanganate lotion. Otherwise nitrate of silver should be applied at this, or in any case at the next sitting. Fused on a probe or aluminum wire or glass rod, the caustic can be applied with exactitude. Schuster recommends Volkmann's spoons to remove necrosed tissue in obstinate ulceration, and if very carefully done the scraping is beneficial. Should small polypi be present, they can be removed in the same way. After the curette the ulcer should be covered with iodol or other substitute for iodoform. When necrosis has taken place, the curette is more decidedly indicated. The instrument should be employed with great care on account of the danger of hemorrhage if it reach the normal tissues which are here so vascular. Mackenzie mentions a fatal case of this kind. Loose pieces of dead bone should be removed with forceps. A large sequestrum, provided it be perfectly loose, may be crushed or divided by forceps or snare, but if still attached it is wiser to wait until separation is completed; that is not likely to be long. Detachment by force is decidedly dangerous. In some urgent cases Rouge's operation may be called for in order to gain access to the seat of the disease, but I have not had occasion to resort to it.

Some deformities may be partially concealed by an artificial nose. In others a rhinoplastic operation may be undertaken.

Hereditary Syphilis.

This manifests itself very early, sometimes within a week of birth. It also occurs at a later period of childhood. In either case it passes rapidly through the earlier stages but later on shows no tendency to subside unless under the influence of thorough antisyphilitic treatment. The earliest manifestation is in the form of acute rhinitis, which the parents often look upon as only a cold. The discharge is at first thin but soon becomes muco-purulent. It is irritant, excoriating the margins of the nostrils and the upper lip. Considerable nasal obstruction is caused by the swelling of the membrane and the accumulation of the secretion so that the child's breathing becomes impeded and noisy, constituting the so-called "snuffles." The general appearance of the child usually excites suspicion, but before the well-known cachexia appears diagnosis may be very difficult, depending on the history and circumstances. Every case of coryza in the first few months of life should be carefully scrutinized. In the majority of syphilitic cases a characteristic eruption soon appears and should always be looked for. The wizened, aged look of the weak, emaciated infant has often been commented upon but is not always present early enough to assist. Mucous patches sometimes appear at the corners of the mouth, but much more frequently about the anus. Sudden cessation of the discharge, with rigors and increased temperature, point to septicæmia, of which, as already stated, H. Weber has recorded the post-mortem proof. Convulsions and other nerve complications may also occur. Possibly these may be sometimes reflex symptoms set up by the blocking of the passage with retained secretions.

The *treatment* of infantile syphilis is essentially mercurial, but local measures are of considerable service. The lip and nasal margins should be protected by cold cream or a simple ointment and an effort made to maintain the patency of the passage. This can often be accomplished by antiseptic sprays. The child's nose may be artificially blown for it, so to say, by a rubber bag and tube fitting the nostril. On compressing the bag the air passes out of the other nostril, forcing before it the accumulation. A pledget-laden probe may sometimes be tried or a suitably made very soft brush. But sprays if persevered in will usually suffice. They should be weakly alkaline. When the passages have been cleared, a weak cocaine spray may be used to contract the tissues and so afford further relief

to the respiration. Spraying or syringing the nares of a child may not be considered an easy task, but patience will almost always overcome the difficulties. If none of the plans named succeed, so necessary is the cleansing that it would be justifiable to plug the nasopharynx during the process by a sponge tampon attached to a wire bent to a suitable form. When ulceration sets in, insufflations of iodoform should be used after each cleansing. If the cartilages and bones become involved, the principles of treatment applicable to adults need only be modified so far as the tender age of the patient renders necessary. With every care the child will probably bear through life the mark of its inheritance.

Nasal Tuberculosis.

Tubercular disease of the nasal passages is very rare and seems to have been unnoticed until 1877 when Laveran (*Union Médicale*) drew attention to it and reported two cases. One of these was confirmed by post-mortem examination but the other seems doubtful. Only between twenty and thirty cases altogether have been recorded and some of these must certainly be excluded. Cartaz (*La France Médicale*) reported a case in 1887 and analyzed those recorded up to that time. No doubt cases have been overlooked, as the nasal affection has generally been secondary and has not added to the patient's distress or danger. In two or three of the cases, however, no signs of pulmonary or other form of tuberculosis were detected until a considerable period after the nasal deposit was observed. No cases should be admitted unless bacilli are found, and in such a position as to exclude surface contamination.

Nasal tuberculosis is met with in two forms—ulcerative and nodular. The disease may attack any part of the membrane, but the floor or the septum seems the favorite seat. The ulcers have the whitish-gray color and irregularly rounded shape without noticeable loss of tissue, just as seen in tuberculous ulceration of other mucous membranes. The secretion is rarely purulent. In the nodular form the deposit presents tumors in the membrane, varying in size from that of a millet seed to half a pea. When they undergo cheesy degeneration the membrane over them softens and ulcerates. Sometimes the nodules resemble papillomata but are usually smaller, flatter, rounder, and have a broad base. Both forms excite the nasal membrane to increased secretion. Hemorrhage occasionally is noticed, more frequently in the ulcerative form. In some cases the tumors have attained a larger size and given rise to obstruction, though this symptom may also be caused by inspissated and retained secretions.

Pain is rarely felt. The diseased part is not exposed to much irritation and the progress is very slow. Usually, therefore, it is a comparatively unimportant complication of more serious tubercular disease.

Diagnosis must rest on microscopical examination. The disease most likely to be mistaken for tubercle is lupus and there is some resemblance to sarcoma. The presence of tuberculosis in some other organ would naturally suggest that chronic nodules or ulcers in the nose might be of the same character. The possible coexistence of syphilis and tuberculosis should also be weighed.

Prognosis is in one sense favorable. The lesion does not increase the gravity of the affection to which it is secondary, but is itself difficult to eradicate and likely to recur.

Treatment.—Antiseptic and disinfectant sprays and washes should be first employed. They may be followed by insufflations of iodol or other powder. These are often the only measures called for. Cure can be hoped for only by total extirpation. Thorough scraping of ulcers, removal of tumors by snare or curette, followed by caustics, have been successful for a time. The wounds heal rapidly, but recurrence has usually taken place in a few weeks, though it may be delayed for several months.

Lupus.

Lupus has already been described among the external diseases of the nose (p. 6), but as it occasionally begins within, and when there is no sign of it on the skin, a few words may be appropriate here in respect to the diagnosis. Billroth has stated that in such a case a mistake is not only "pardonable but unavoidable." This remark is in reference to syphilis but is equally applicable to some cases of tuberculosis. In its early stage it is also extremely difficult to distinguish lupus from epithelioma. Sarcoma, too, may be named as a possible source of error. The history, circumstances, and the effects of treatment furnish valuable evidence, and the course of the disease will remove many obscurities, but the microscope must be called in definitely to decide the diagnosis.

Nasal Diphtheria.

This is usually due to the exudative inflammation extending from the pharynx, but sometimes the disease commences in the nasal membrane. It then appears at first as a simple rhinitis but soon puts on graver features. There is an irritating ichorous discharge which excoriates the skin, followed by the appearance of more or less false

membrane on the turbinated bodies and the septum, but this may be seen only by the rhinoscope, as it is generally more abundant at the posterior orifices. Septic absorption is apt to ensue if the discharge be allowed to accumulate and decompose in the passages, which should therefore be cleansed by gentle syringing or spraying with disinfectant lotions. Epistaxis is common but may be met by astringent lotions or insufflations. Plugging is contra-indicated.

Infectious Diseases.

The nasal passages suffer more or less in the exanthemata, and other fevers, but their condition is often unnoticed in the presence of the general symptoms.

Measles is usually ushered in by a watery discharge, which is sometimes followed by epistaxis or by severe inflammation. Occasionally atrophic rhinitis seems to be started by an attack of measles.

In *scarlet fever* also there may be only a mild catarrh, though in other cases there is severe rhinitis with great swelling of the membrane and an irritating discharge. Ulceration may also ensue and give rise to epistaxis.

In *small-pox* pustules may form in the nasal passages and give rise to obstruction and to bleeding. At a later period union of the raw surfaces left by the pustules on the outer and inner walls occurs and may occlude either or both nostrils.

Typhoid Fever.—In this disease the mucous membrane is dry and parched. Sometimes this is followed by ulceration which spreads and proves rather intractable. Necrosis of the septum may occur, followed by perforation. In other fevers similar lesions may be observed.

Glanders is the subject of an article in a later volume of this work, to which the reader is referred.

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DISEASES

OF THE

ACCESSORY SINUSES OF THE NOSE.

BY

JONATHAN WRIGHT,

BROOKLYN.

DISEASES OF THE ACCESSORY SINUSES OF THE NOSE.

Rhinology is too recent a subdivision of medical study to permit yet of much crystallization of thought. The still more recent investigations made into the subject of the diseases of the accessory sinuses of the nose do not permit in this place of any attempt at presenting the consensus of opinion of rhinologists on the subject, nor of any critical remarks on their works, nor do they warrant any dogmatism that may unconsciously appear in the expression of the views of the writer.

By the accessory cavities of the nose we understand certain recesses hollowed out of the neighboring bones and communicating solely and more or less directly with the nasal chambers proper. These are the maxillary, frontal, ethmoidal, and sphenoidal sinuses.

Rather than enter into any long anatomical description of them I have preferred to insert drawings of specimens from my collection which will serve to remind the reader of the general topography, and will illustrate some points referred to in the description of the diseases of the parts.

In Fig. 17 I have attempted to show by means of probes *P* and *P'* how fluid coming from the frontal sinus through the fronto-nasal canal may enter the nose at the upper end of the gutter of the hiatus semilunaris, and may trickle down along that deep groove and enter the ostium maxillare without appearing in the nasal chambers until the maxillary antrum is full; and then all symptoms may be identical with those of an original antrum affection. Opening and draining the maxillary antrum will fail to arrest the flow of pus in a suppurative case. In this drawing it will be seen that the middle turbinated bone has been entirely removed to show the structures which lie beneath it. A horizontal section has also been made just below the inferior turbinated bone so as to remove the floor of the antrum and show the cavity.

In Fig. 18 a vertical transverse section has been made so as to fall directly through the ostium maxillare, and also expose the ostium

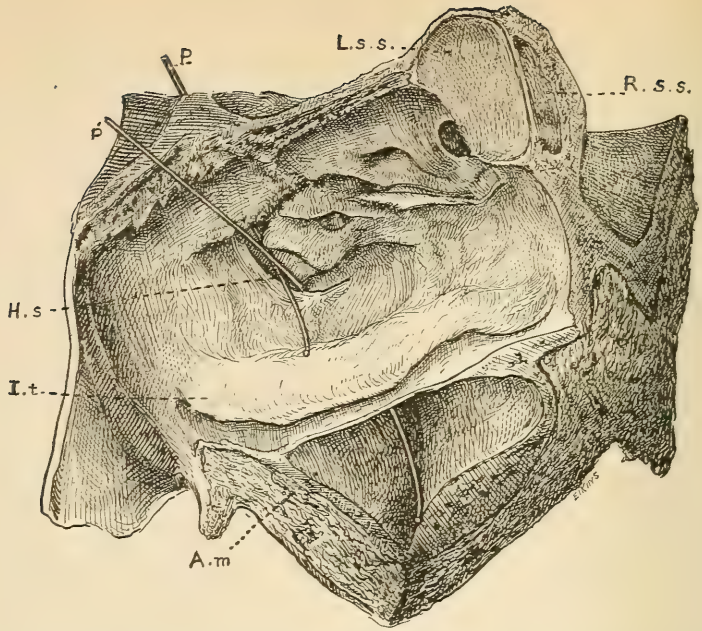


Fig. 17.—Specimen Showing Route Taken by Fluids From the Frontal Sinus to the Antrum. The palatal process of the superior maxilla and the middle turbinated bone have been removed; the specimen is held obliquely.

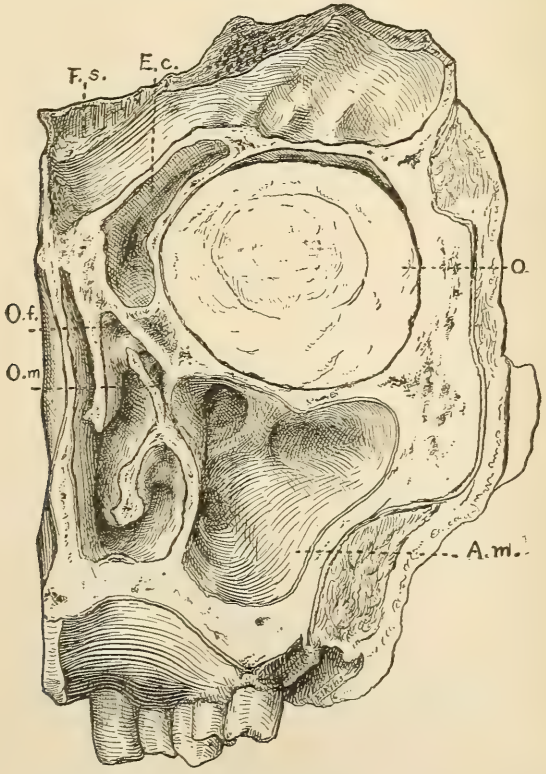


Fig. 18.—Transverse Vertical Section Passing Through the Ostium Maxillare.

frontale. Besides again showing how the maxillary antrum may take the drain from the frontal sinus, it may also be seen how ineffectual any syringing out of the antrum of Highmore must be *per vias naturales*. It also shows the possibility of penetrating through the fronto-nasal canal into the frontal sinus, but the reader must be warned that while this appears feasible in the dissecting-room speci-

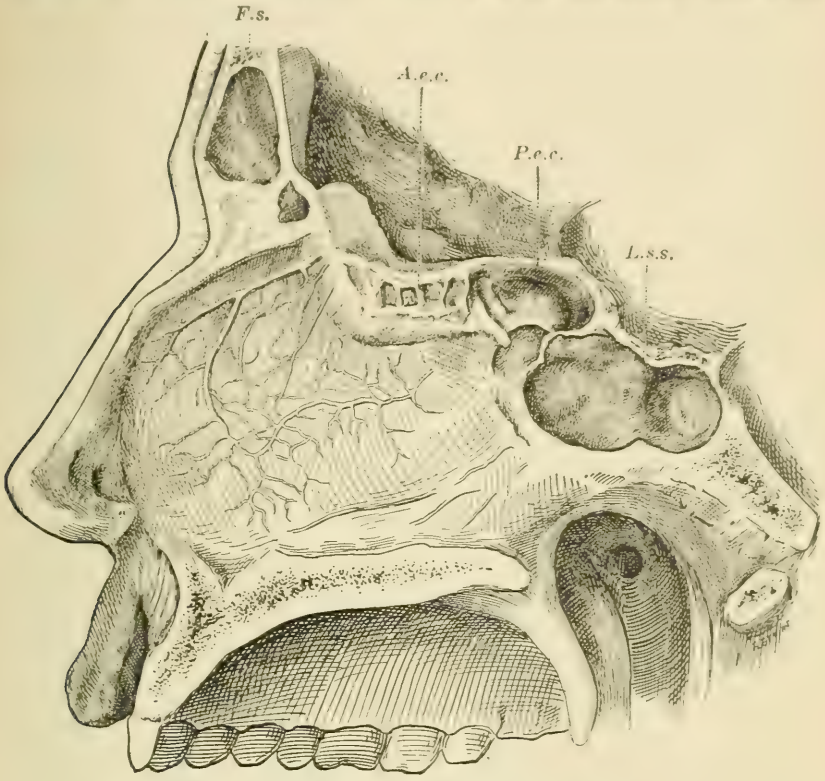


FIG. 19.—Sagittal Section Showing the Left Frontal, Ethmoidal, and Sphenoidal Sinuses.

FIGS. 17-19.—*A.m.*, Antrum maxillare; *O.f.*, ostium frontale; *O.m.*, ostium maxillare; *H.s.*, hiatus semilunaris; *F.s.*, frontal sinus; *E.c.*, ethmoid cell; *A.e.c.*, anterior ethmoid cells; *P.e.c.*, posterior ethmoid cells; *S.s.*, sphenoidal sinus, *L.s.s.*, left sphenoidal sinus; *R.s.s.*, right sphenoidal sinus; *I.t.*, inferior turbinate bone; *O*, orbit; *P*, probe in fronto-nasal canal; *P'*, probe in ostium maxillare and antrum.

men when laid open for inspection, it is practically quite another matter in the living subject.

In Fig. 18 also attention is drawn to the large size of the ethmoidal cell, which is separated from the orbit by the thin plate of bone known as the os planum. In the specimen from which this drawing was taken that cavity, larger than the frontal sinus, also communicated by a small opening with the hiatus semilunaris, so situated that its

secretions would also drain into the maxillary antrum. It will be seen how readily access may be gained to the ethmoidal cells from a curved incision above the inner canthus of the orbit.

By reference to Fig. 19 it will be seen that a differentiation of pus as coming from the posterior ethmoidal cells or from the sphenoidal sinus is impossible. Referring to Fig. 17, the opening of the sphenoidal sinus is seen considerably above the posterior end of the ridge left by the removal of the middle turbinated bone. The vertical section in this specimen (Fig. 17) was made through the left nasal fossa close to the septum, which was then removed; thus the section fell through the left sphenoidal sinus, leaving the right sinus with its opening intact.

The distance from the anterior nasal spine to the sphenoidal sinus differs widely according to different authorities and doubtless differs widely in different cases. The distance from the anterior nasal spine to the anterior wall is reported in some cases to be less than two inches and in some as more than three inches. I have a patient under observation, suffering from excessive atrophic rhinitis, in whom a full view of the opening of the sphenoidal sinus on one side can be had. By measurement it is three inches to the anterior wall and three and one-half inches to the posterior wall of the cavity.

Zuckerkandl apparently does not give us any information on this very practical point, and perhaps it would not be amiss to give some figures obtained from adult heads taken from a dissecting-room and preserved in alcohol. They are four in number and have been sawed longitudinally, and having been separated and used for other purposes, it is impossible to be sure now which halves correspond. Therefore these measurements are for the eight halves of four adult heads. It will be seen how little the variations are. For the anterior nasal spine I have thought best to take not the bony prominence itself but that bony prominence covered by the soft tissues of the columna, such a condition as obtains in life. It must be remembered that with soft, yielding tissues mathematically accurate measurements cannot be made in a rhinoscopic examination.

From the Anterior Nasal Spine

	To anterior wall of sphenoidal sinus.		To posterior wall of sphenoidal sinus.	
	Inches.	cm.	Inches.	mm.
1.	2⅛	5.4	2⅝	6.6
2.	2⅜	6	3	7.6
3.	2½	6.3	3⅛	7.9
4.	2¼	5.7	3¼	8.2
5.	2¼	5.7	2¾	7
6.	2¼	5.7	3	7.6
7.	2½	6.3	3¼	8.2
8.	2⅜	6	3	7.6

It will be readily understood that these measurements, in spite of the pretty uniform figures given here, must vary according as the head is dolicho-cephalic or brachio-cephalic. Four heads are too small a number with which to attempt to establish an average figure and they are given here only in hope that the subject will receive more extensive investigation, as it is a matter of considerable importance to rhinologists in the diagnosis and treatment of sphenoidal sinus disease. Unfortunately, however, the situation of the sinus cavity varies in shape and in its relative position in the bone, and especially in its opening into the nose. In these heads with the mucous membrane shrunk with the alcohol and in the case of atrophic rhinitis referred to above, the openings were 2 to 5 mm. in diameter. Under normal conditions they are much smaller. In these cases the openings were considerably above the level of the middle turbinate bone. The thickness of the bony walls of this sinus varies greatly, there being sometimes only a very thin plate of bone separating the cerebral and sphenoidal cavities, and the same is true for its pharyngeal or nasal wall.

The declaration of Zuckerkandl that "boring into the sinus through the anterior wall is easily done" is the statement of a learned anatomist more familiar with post-mortem privileges than with the practical difficulties of rhinoscopic examinations and intranasal operations.

The posterior ethmoidal cells frequently communicate directly with the sphenoidal sinus. Sometimes they are separated from it only by a thin shell of bone so that any process involving the one is pretty sure to include the other also. Indeed, there seems to be anatomically and pathologically a greater barrier between the anterior and posterior ethmoidal cells than between the latter and the sphenoidal sinus.

Various statements have been advanced in regard to the physiological functions of the accessory sinuses. It has been stated that they are magnifiers or modifiers of the voice and that they reinforce the power of olfaction, but these assertions have not been supported by the majority of investigators. Their existence, of course, renders the weight of the facial bones less than they otherwise would be, and very probably helps to break the force of blows or jars which otherwise would be transmitted more directly to the important ganglia at the base of the brain.

The Antrum of Highmore.

The maxillary sinus or antrum of Highmore is the most easily accessible to exploration and operation and is on that account also the most exposed to injuries. Having in its floor the roots of the teeth,

it is especially liable to disease transmitted from them and from the buccal cavity. Opening by a narrow orifice at the top and having a large dependent cavity, it is so situated as not only to receive the drain from ethmoidal or frontal sinus disease, but opening at the hiatus semilunaris it is very liable, in polypoid disease of the nose, and even in simple hypertrophies, to be occluded by adventitious growths. The mucous membrane lining it is not so apt as that in the ethmoidal sinus to become inflamed by continuity when the nasal mucosa is affected in severe attacks of coryza, but nevertheless this frequently occurs. In view of all these facts, we may easily understand why our knowledge in regard to antrum disease is older, more extensive, and more accurate than is the case with the other accessory sinuses.

Acute Catarrhal Inflammation.

Of the acute catarrhal inflammations of the mucous membrane of the antrum we know little or nothing. It is reasonable to suppose that they occur with considerable frequency in connection with the severe forms of coryza. If so, they usually subside spontaneously and pass undetected. Sometimes, however, these attacks evidently are the starting-point of a purulent inflammation.

Acute Purulent Inflammation.

Acute purulent inflammation of the antrum may arise thus during the course of a coryza because the ostium maxillare is temporarily occluded. The retained secretion becomes a good culture medium for pyogenic bacteria. The constant presence of pyogenic bacteria in the healthy nose and its accessory sinuses is, at the present writing, a matter of some doubt, but however that may be, it evidently requires something more than their mere presence to excite suppurative action. The staphylococcus pyogenes, the streptococcus, and Friedländer's bacillus with other forms have been found in the pus.

Retention of secretion during nasal inflammations, the spread of purulent inflammation from a tooth root, injury to the bony walls from blows or penetrating wounds are all of them causes of acute suppurative inflammation of the antrum. These may be the causes of an acute inflammation and that may or may not go on to a chronic form. It probably does not become chronic except from one of two conditions—the permanent occlusion of the ostium maxillare, or the continued presence of a source of infection or irritation such as a carious tooth root, a foreign body, or carious bone, or fetid secretions from

purulent ethmoidal or frontal sinus disease which may drip from above through the hiatus semilunaris and the ostium maxillare. Permanent occlusion may be due to a congenital closure of the ostium maxillare or to adventitious nasal growths. The acute cases, that is, those which recover spontaneously in a short time, are most frequently those which occur during a coryza. The chronic cases in my experience more frequently arise from dental caries than from any other cause, or indeed than from all other causes combined.

Accessory sinus disease rarely occurs in children, and this is especially so in the case of the maxillary antrum, partly because the sinuses are not well developed until after puberty, but principally, I think, because children rarely suffer from the chronic obstructive nasal diseases, and also because the roots of the molar teeth do not penetrate the floor of the antrum. Nevertheless a case has lately been reported by M. Rudaux in the *Annales des Maladies de l'Oreille et du Larynx* for September, 1895, in which the presence of a little molar tooth in the antrum of an infant three weeks old was the cause of a profuse and serious suppuration. A few other cases in children are also on record (see Seifert's paper in the *Revue de Laryngologie, d'Otologie et de Rhinologie*, No. 21, 1895).

Symptoms.

The most striking symptom is usually an abundant unilateral discharge of pus from the nostril, either continuously or at intervals. If at intervals there is considerable relief, after the discharge, from the pain and the feeling of heaviness in the nose and head generally.

Sometimes the pain is very severe and the discharge may be absent altogether, or very scanty, indicating some obstruction at the ostium maxillare. There may be considerable systemic disturbance, fever, chilliness, headache, malaise, etc. At the end of a few days, with or without simple treatment directed to the coryza, these cases recover, and they may never have another attack. I have seen this course taken even by an acute empyema of dental origin.

Rhinoscopic examination shows pus in the middle meatus coming from under the turbinated bone. There is usually considerable tenderness to pressure over the antrum. In some cases, the ostium maxillare being occluded, pus is not discharged by the nose. Where there is no history of a purulent discharge, the tenderness to pressure is marked, and the distention of the cavity may be such as to cause a bulging of its bony walls, producing nasal stenosis, exophthalmos, external swelling of the cheek, or even a distortion of the buccal arch.

Differential Diagnosis.

When this swelling of the antrum walls does not betray the seat of the trouble, the rhinologist frequently has a difficult problem to solve, for not only must he settle the question of pus in the maxillary antrum, but he must attempt to decide whether there is not also involvement of the other accessory sinuses.

A persistent unilateral nasal discharge in a child, especially a young child, nearly always means that there is a foreign body in the nose. In an adult it nearly always indicates a purulent inflammation of one of the accessory sinuses.

Anterior rhinoscopy shows that the purulent discharge apparently comes from under the middle turbinated bone. This, however, must be determined by carefully wiping and washing away what is seen in the nostril, as frequently pus may be seen above the middle turbinated bone and between it and the septum. Why this should so frequently appear it is hard to explain, for the opening from the antrum is under the middle turbinated bone. We must suppose part of the pus to pursue a tortuous course as a result of capillary attraction from the apposition of two mucous surfaces.

Usually when the maxillary sinus is alone affected the pus, once washed carefully away from the visible parts of the nasal chambers, does not quickly reappear, as it nearly always does when the frontal or ethmoidal sinuses are involved. When it does not reappear the patient's head should be lowered so that the antrum is turned upside down. Having held the patient's head in this position a short time, a reinspection will usually show that pus has again oozed into the nose from under the middle turbinated bone.

Czermak showed in 1858 that a strong light could be made to penetrate the larynx from the outside and be seen in the laryngoscopic mirror, illuminating the laryngeal cavity and tissues, throwing shadows differing in intensity according to their translucency. It had been mentioned occasionally in laryngological text-books, but being of no practical value had been almost forgotten until the invention of the Edison incandescent electric lamp, when Voltolini again experimented with it in the larynx and Heryng and Vohsen applied it to the investigation of the diseases of the antrum of Highmore. Here it has proved of very great value, though it is not by any means to be implicitly relied upon.

The patient is examined in a dark room. The small electric lamp such as shown in Fig. 20 is put in his mouth. He is directed to close his lips around the stem. The current is turned on and the patient's face is illuminated by the transmitted light. The infraorbital

region on the affected side will be seen to be less bright than on the unobstructed side, the pus in the antrum being more or less opaque.

The patient on shutting his eyes will notice that less of the transmitted light penetrates the eye of the affected side. The physician

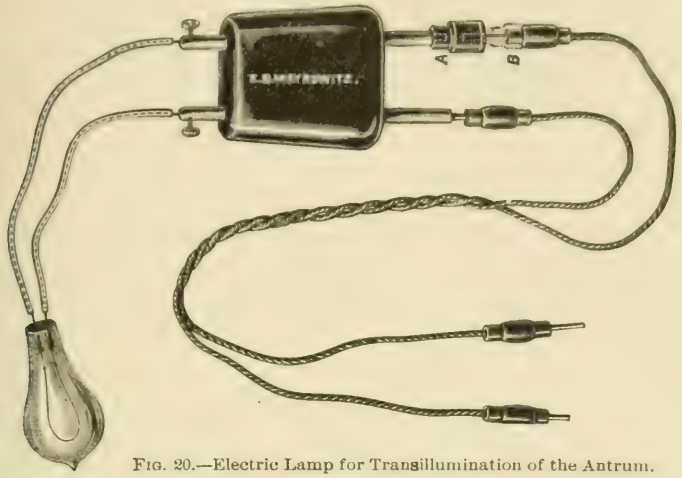


Fig. 20.—Electric Lamp for Transillumination of the Antrum.

may sometimes also observe with the ophthalmoscope that less light enters that eyeball.

There are some conditions which may interfere with this test. There may be unequal thickness of the bony or the soft tissues of the parts, bony spurs or deviations of the septum or turbinal hypertrophies, or, when the trouble is bilateral and there is a lack of compari-

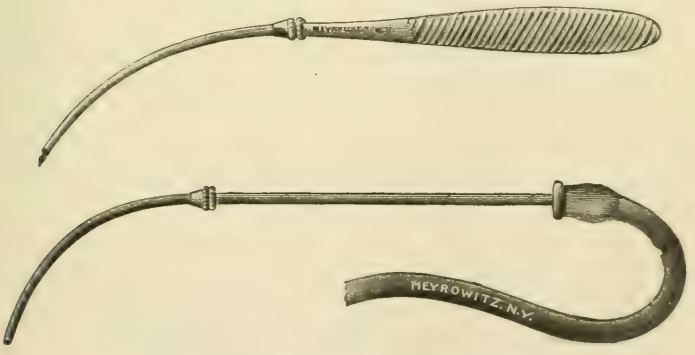


Fig. 21.—Trocar and Cannula of Dr. Myles.

son, transillumination may fail to be of value. In many cases, however, the evidence is so striking that, combined with other symptoms, the diagnosis becomes a matter of considerable certainty. Where

any doubt is still felt, puncture of the antrum from the inferior meatus with Krause's trocar and cannula, or with a similar instrument used by Dr. Myles and shown in Fig. 21, the less firm and resisting wall in the middle meatus may be transfixed and the pus washed from the antrum by a boric-acid solution introduced through the cannula and escaping with the pus by the natural opening at the hiatus semilunaris. Under cocaine this causes very little pain, but in acute cases it is perhaps better to wait until it becomes evident that surgical procedure and therefore an exact and certain diagnosis are necessities.

Treatment.

It is impossible and undesirable in a work of this kind to relate the history or describe all the procedures for treating the diseases of the maxillary sinus, and only those which have proved most serviceable in the experience of the writer will be mentioned. In acute cases the treatment is comparatively simple.

The nasal chambers should be thoroughly cleansed with a warm alkaline solution, used by the physician in a post-nasal syringe or by the patient in a properly constructed and cautiously used douche cup. This treatment should be continued if necessary for several weeks. Any obstruction to the outflow of matter should be removed from the nasal chambers, especially in the neighborhood of the middle meatus. Hypertrophies should be cauterized, polypi removed with the snare, spurs sawed from the septum, and the latter straightened if necessary. I believe that all the acute cases of suppuration of the antrum not due to defective teeth, foreign bodies, or injuries, would recover under the above treatment were it promptly begun and thoroughly carried out. I have known a case to recover with simple douching when the trouble was caused by inflammation from a tooth filling. Occasionally, however, these cases beginning with a coryza only partially subside of themselves, obstructions in the nose are not removed, and the discharge, which has become scanty though persistent, is unnoticed or disregarded, until so much time has elapsed that a pyogenic membrane has been formed within the antrum; the cases which have become thus chronic require a more or less vigorous treatment directed to the antrum cavity itself. On the other hand pain and systemic disturbance may in some of these acute cases render immediate opening of the antrum necessary.

Chronic Purulent Inflammation.

In chronic empyema of the antrum of Highmore we must distinguish between those cases in which there is free communication with

the nose and those rarer ones in which the ostium maxillare is occluded—the so-called latent cases.

Empyema with Patent Ostium Maxillare.—The amount of discharge varies. In the very old cases—and their histories may run back many years—the discharge is usually small in amount and finds its way into the nose at comparatively long intervals, perhaps once a day or once in two days. Usually it is more abundant than this. Whether abundant or scanty, however, there is apt to be a history of the discharge taking place more copiously on a change in the position of the head. When the patient lies down it may run out into the nose and down the throat; or what has collected in the nasal chambers during the night finds its way into the pharynx when the patient arises in the morning. The sufferers usually complain of a more or less fetid odor and an acrid, bitter taste, or there may be simply a sweetish taste to the discharge. Pain in these unobstructed cases is frequently not a prominent symptom. In those cases, however, in which there is some obstruction of the ostium maxillare and sometimes where there is no evidence of it, the pain may be severe and referred to the infraorbital region, the bridge of the nose, or even the supraorbital region, and it may radiate in various directions, or there may be a dull general headache. Very frequently, even when there is a free vent to the pus, we may have a narrowing of the field of vision on the affected side, congestion of the fundus of the eye, or conjunctivitis.

The general health of these patients is frequently affected. They become depressed in spirits and their life is a burden. They are conscious of the offensive smell and think their presence is annoying to those about them, though as a matter of fact the odor is rarely appreciated by others, this being quite contrary to the rule with ozæna.

Rhinoscopic examination usually shows the presence of pus in the middle meatus, but it may be absent not only at the first examination, but also at subsequent ones, and the statements of the patient must be relied on in regard to it. Chronic empyema of the antrum is very apt to be associated with intranasal œdematous polypi, and the antrum itself may be filled with this so-called polypoid degeneration of the mucous membrane. Without attempting here to go into a discussion of the subject, I may state my belief that usually the intranasal polypi are a result and not the cause of the antrum suppuration, and that the presence of foul pus in the antrum is the cause of its mucous membrane undergoing the inflammatory changes which result in the formation of this loose œdematous tissue. It is convenient to give them the name of polypi or fibromata when they are pedunculated, but exactly the same histological condition of the mu-

cous membrane of the nose and antrum is found with no tendency to the formation of pendent tumors. Usually drainage of the antrum from below promptly cures the polypoid condition of the nose, while removal of intranasal polypi rarely if ever cures the coexisting antrum suppuration, and the polypi themselves quickly recur as long as the pus bathes the nasal mucosa.

Latent Empyema.—A large number of cases have been reported, especially in Germany, of latent empyema of the maxillary antrum, *i.e.*, the opening of the antrum having become occluded, there is no external discharge of pus, but the patient suffers from infraorbital or supraorbital pain, hemicrania, reflex ocular symptoms, or vasomotor intranasal symptoms such as watery discharge from the nostrils, occlusion from turbinal congestion, etc.

Examination by transillumination or by puncture reveals the presence of pus in the antrum.

Duration and Course.

As has been said, suppuration of the antrum may extend over many years. From the fact that so many cases are now discovered while formerly so few presented themselves for treatment, we must surmise that many of those cases eventually recover of themselves without treatment. There have of late been reports of cases that, refusing operation, eventually recovered entirely, after a long-continued and gradually diminishing discharge. I myself know of one such case where the trouble arose from injury. Nevertheless these are always cases for surgical interference, even though the results are not uniformly successful, because they suffer great inconvenience and distress, and because spontaneous cure is doubtful, and even if it occurs is usually a matter of years.

Treatment.

When the discharge has lasted for several months we must consider the choice of site for opening and draining the sinus.

It has been opened at almost every possible point and almost every site has had its firm advocate. As said above, description or even mention of all these operations cannot be made here. We should first consider the natural opening at the hiatus semilunaris. In the vast majority of cases, notwithstanding the statements of various operators to the contrary, I am convinced that it is impossible to penetrate through the ostium maxillare into the true cavity of the antrum from the anterior nares during life, with any instrument, however ingeniously bent or however skilfully manipulated. It is doubtless possible to reach the gutter of the hiatus semilunaris and

get solutions into the antrum in many cases, but the efficient flushing of the antrum can be attained in the vast majority of the cases only by breaking down or penetrating through the lip of the opening. In those few cases in which a bent cannula can be introduced under the middle turbinated bone and access to the antrum be readily gained through the ostium maxillare, persistent attempts should be made to cure the case by syringing out the cavity daily with warm boric-acid solution. A twenty-per-cent. solution of the ten-volume peroxide of hydrogen should also be occasionally used. From the fact that there are some cases in which the anatomical arrangement is such as to allow this procedure it should perhaps be attempted in all cases. If, however, the antrum walls are to be pierced, an opening should be made at a more accessible point and through the most dependent portion where thorough drainage can be kept up. If there are carious tooth roots in the upper jaw of that side which project into the antrum, they should always be extracted and the perforation made through one or more of their alveoli, for we thus not only drain the antrum at a suitable point, but at the same time remove the primary cause of the trouble. The two front molars and the first pre-molar project more frequently than others, but any of the teeth lying between the incisors and the wisdom tooth may, by having their roots inflamed, become the starting-point of abscess of the antrum. Usually their roots are covered by a thin lamella of bone, but this is not always the case. When the tooth is extracted, sometimes a flow of pus immediately follows and the alveolus of the tooth is found to communicate freely with the antrum. A strong hand drill is usually necessary, however, to break through the bony floor, and this will be found more efficacious and trustworthy than the electric drill or trephine.

With a warm boric-acid or other cleansing solution the antrum should be thoroughly flushed out. Attempts may be made to find a

Fig. 22.—Bosworth's Antrum Tube.

foreign body or ascertain the condition of the antrum with a probe. As a rule, however, very little can be learned by the way of a perforated alveolus either by probing or by illumination. If there is a sound tooth near enough to the perforation one of Bosworth's antrum tubes (Fig. 22), of as large a size as possible, may be inserted and wired fast, as otherwise the perforation will usually close in a day or two. This should be kept in for a fortnight and the antrum should be thoroughly flushed out several times daily with boric-acid solution and occasionally with peroxide of hydrogen, 1 : 5 of a ten-volume solution.

After a day or two the secretion will usually be very much abated. If the disease is confined to the antrum pus will, as a rule, no longer be seen in the nasal chambers. At the end of two weeks there will, even if the case is going to recover, still be a little discharge from the antrum because of the presence of the tube. During meals a small

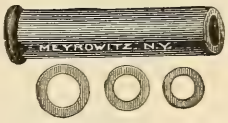


Fig. 23.—Myles' Soft-Rubber Antrum Tube.

cork should be put in the mouth of the tube to prevent the entrance of food. If there are no teeth to hold an antrum tube one of Dr. Myles' soft-rubber tubes may be used (Fig. 23).

At the end of two weeks the tube may be withdrawn and the perforation allowed to heal. If simple drainage and washing out the antrum are going to conquer the trouble at all they will do it in that time.

When syringing through the hiatus semilunaris has been ineffectual or impossible, or when the operation through the alveolus has failed, or when there are no carious teeth to remove from the upper jaw, and even when the patient has *no* teeth, the preferable site for opening the antrum seems to me to be in the canine fossa just above the alveolar border.

The patient is thoroughly etherized and a short vertical incision is made through the mucous membrane and periosteum just posterior to the canine eminence. This is joined at its centre by a horizontal incision, parallel with the alveolar process, and running as far back as the malar process. The periosteum is elevated as quickly as possible, which helps to check the rather free hemorrhage. With a small chisel or with an electric trephine a fenestra is cut in the bone. The mucous membrane of the antrum presents in the opening, and is incised freely, which usually causes considerable obstinate bleeding. This, however, may be checked by rapidly packing the antrum with iodoform gauze. As a rule, very little pus escapes by the incision. The position of the patient on his back causes the pus to run out of the natural opening into the nose as soon as a vent hole is made by perforation into the opposite wall of the antrum. As soon as hemorrhage has ceased the packing is removed and with a pair of small bone forceps a large enough opening is made in the bone to allow of the easy admission of the index finger. The antrum is thoroughly washed out, explored with the finger, and illuminated by reflected light. Any pyogenic membrane or crumbling bone is thoroughly

scraped with a sharp curette. Sometimes bony septa divide the antrum. These, if extensive enough to interfere with the thorough exploration of the cavity and subsequent packing, are broken down with drill or bone forceps. Care should be taken to explore the antrum near its nasal opening. The cavity should be then firmly packed with long strips of iodoform or borated or bichloride gauze, care being taken to get the gauze well up to the remote corners of the cavity. This should be left *in situ* for forty-eight hours, the patient being kept in bed. At the end of that time it is to be removed and careful inspection and exploration again made of the antrum, as frequently more can be learned when the antrum is dry and clean than at the time of the operation. If the patient's general condition allows of it, as it usually does, he may now get up. The antrum is again thoroughly washed out and packed, and this time the gauze may be left in three or four days; subsequently it may be possible to leave the packing in for a week. After three or four weeks' time, no dead bone or other complication having been discovered, the packing may be permanently removed and the patient be directed to syringe the antrum thoroughly after each meal until the opening closes up, which it will do in a surprisingly short time. Unfortunately even this thorough procedure fails to arrest the suppuration in some cases, and after the opening has healed pus again appears in the nose. There is nothing to do, under such circumstances, but to open up the cavity again, repack it for a time, and then have a dentist fit a vulcanite obturator to the opening under the cheek, so that the antrum may be thoroughly cleansed daily.

This operation, as a rule, produces no appreciable sinking or deformity of the cheek if care is taken not to remove the bone to any greater extent in a vertical direction than is necessary to admit the finger. It may be more freely removed in a direction parallel with the alveolar border.

Mucocele.

Cases have been reported from time to time of serous accumulations in the antrum of Highmore. Noltenius (*Monatschrift für Ohrenheilkunde*, April, 1895) has lately reported thirty-seven cases, several of them bilateral.

Their pathology is not understood. The symptoms they present are very indefinite, being chiefly sensations of pain and heaviness in the head, and the ordinary symptoms of chronic rhinitis. There is an intermittent watery discharge from the nose.

Occasionally the watery discharge is more or less constant. This symptom, as indeed also the pain, apparently depend upon the de-

gree of permeability of the ostium maxillare. A satisfactory diagnosis can be made only by puncture with the trocar and cannula.

Draining and washing out the antrum seem usually to have been sufficient to cure this condition.

Notwithstanding the large number of cases reported by the author referred to, and occasional reports by others, these cases seem to be, at least in this country, of very rare occurrence.

Ozæna.

A number of cases of ozæna of the antrum have also been reported. Crusts with the characteristic odor are found on opening the cavity. This may accompany a similar condition in the nasal chambers proper or may exist separately. It may possibly be the explanation of those rather rare cases of ozæna in which there is no crust formation in the nose, but a persistent characteristic odor (W. Robertson, *The Lancet,* April 29th, 1893).

Tumors.

Under this head we may mention the so-called myxomatous polypi, but only, as said above, to exclude them from the category of true tumors. This Chiari showed in his "Erfahrungen auf dem Gebiete der Hals- und Nasen-Krankheiten," p. 71, and it has been dwelt on to some length by the writer (*New York Medical Journal,* November 4th, 1893).

Cysts occasionally form in the mucous membrane of the antrum by the closure of one of the glandular conduits, and fill the whole cavity. A case has recently been reported by Dr. C. H. Knight, in which the cyst filled the antrum and, pushing out the anterior bony wall, presented itself above the alveolar border. Dentigerous cysts have also been reported in this cavity. Bony cysts springing from the teeth, exostoses, and bony tumors of the walls of the antrum have been described and figured by Zuckerkandl as post-mortem findings. They rarely come to notice during life.

Other benign tumors of the antrum are very rare. While I have seen several cases of primary carcinoma and sarcoma starting within the antrum of Highmore, I have never seen during life a fibroma or an osteoma or a true myxoma of this region. Malignant disease of the antrum belongs to that division of general surgery which treats of diseases of the jaws, and has found such an able exponent in Sir Christopher Heath.

Foreign bodies such as teeth, spicula of bone, drainage tubes, probes, insects, etc., have been found in the antrum, but require no

notice here, as they are usually not detected until the cavity is thoroughly explored by some operation, such as detailed above, for the resulting suppuration.

The Frontal Sinus.

Acute Catarrhal Inflammation.

This is a very frequent and painful complication of a similar inflammation of the nasal mucosa. The frontal sinus is a smaller cavity and has a longer and narrower outlet than has the maxillary sinus. Any inflammation, therefore, more readily causes pressure by the apposition of the swollen mucous membrane, and the outlet being more easily occluded, retention of secretion is more frequent. These conditions give rise to symptoms which allow of their recognition very much more frequently than is the case with the other accessory sinuses, but their occurrence is probably in reality no more frequent in attacks of coryza. On the other hand, its dependent outlet into the nose, and the absence of the frequent cause of suppuration of the antrum, viz., carious teeth, both tend to render empyema of the frontal sinus less frequent than that of the antrum.

Symptoms.

In the course of a coryza, as was noticed especially during the late epidemics of influenza, the patient begins to suffer from intense pain in the frontal region, usually bilateral. This may arise at the beginning of the attack, with the other symptoms of a coryza, or it may be delayed until the later stages and even appear after the entire subsidence of the intranasal inflammation.

The eyes are usually affected, there being considerable photophobia and conjunctival congestion. There may even be slight œdema of the connective tissue around the eyes.

Firm pressure with the finger under the superciliary ridge causes considerable pain. This condition may last several days or even weeks. It is apt to pass off quite suddenly with an abundant flow of mucus or of a muco-purulent discharge.

Gastro-intestinal causes may give rise to some at least of these symptoms, and indeed gastro-intestinal disturbance is frequently noted in this form of coryza.

Usually these patients do not seek treatment, considering the attack an ordinary one of coryza. Occasionally, however, the pain and discomfort are so severe that they do ask for aid.

Treatment.

Pain is usually the principal symptom for which the patients seek relief. It is well to correct any intestinal trouble by a brisk cathartic — ten grains of blue mass. The pain can usually be controlled by the administration every hour of a pill containing camphor, gr. $\frac{1}{2}$, opium, gr. $\frac{1}{10}$. A similar presciption is put up in the form of a tablet containing also a little belladonna and aconite and known as coryza tablets. In addition these patients occasionally obtain the greatest relief from intranasal douches of hot water. A fountain syringe is filled with hot water in which is dissolved a little salt (one-half per cent.) and with the nozzle so directed that the stream will reach the region of the anterior end of the middle turbinated bone a quart or more is allowed to run through at a temperature as high as the patient can bear with comfort. The head should be bowed forward so that the water will run out freely anteriorly and not back into the naso-pharynx. Heat may be applied externally by means of hot-water bags.

Frequently, however, nothing gives relief to these patients but opium given as above indicated or in the form of Dover's powder. A Turkish bath will sometimes give great relief, but it should be taken very cautiously and absolutely no exposure of the surface to cold should be permitted for some time afterwards. Confinement to a warm room must be insisted upon.

Acute Purulent Inflammation.

Empyema of the frontal sinuses is said to be much more frequent on the right side than on the left. At any time during the involvement of the frontal sinuses in a coryza the secretion may become purulent and abundant. All the symptoms mentioned above, usually in an aggravated form, are present. In addition pus will be found in the middle meatus, usually high up and well forward. The differential diagnostic points of maxillary sinus suppuration have been dwelt on at length. Transillumination of the frontal sinus is a matter of some difficulty and does not give reliable results, and there is in fact no satisfactory way of diagnosticating acute frontal sinus suppuration from that of the ethmoid cells, as the rational signs are not reliable. A frontal sinus suppuration may persist for several weeks and then subside of itself. While there is acute inflammation within the nose the same plan of treatment should be adopted as for the catarrhal form. When this has subsided a little and the suppuration persists, the middle meatus must be cleared of any obstruction that may exist and the nose must be kept thoroughly clean for several weeks.

Chronic Purulent Inflammation.

This almost always has its origin in one of the acute attacks or perhaps is the result of repeated attacks. The various forms of nasal obstruction, and especially that form of ethmoid disease which results in hypertrophy of the anterior end of the middle turbinated bone, nasal polypi, deviations of and spurs upon the septum, together with general hypertrophy of the nasal mucous membrane, are intranasal conditions which favor the supervention of a chronic purulent inflammation of the frontal sinus upon what would otherwise be an ordinary coryza.

The writer is very far from urging every one with a crooked nasal septum, or with a spur on it, to place himself in the hands of a rhinologist for operation on that account alone; but the occurrence of frequent, protracted, and painful coryzas in a patient with these conditions is an indication not to be disregarded. This same warning applies of course to all accessory sinus troubles.

Symptoms.

The distinctive symptoms are those of pain and tenderness in the region of the frontal sinuses. There may be some bulging of the walls of the frontal sinus, impinging on the contents of the orbit or appearing at the inner angle of the orbit, or even, as I have seen it, on the anterior surface of the frontal bone. Such evidence as this, with or without pus in the nose, is conclusive.

The pain may be as severe as in the acute form, but is more apt to be of a dull character and pretty constant, with exacerbations in damp weather and on taking cold. The pus discharge may be scanty but constant, or it may collect in considerable quantity, causing great pain, and then gush forth into the nose with considerable relief from the suffering for a time. The patients soon become apathetic and depressed in spirits, frequently forgetful, and unable to attend to business matters. When frontal sinus trouble exists alone, the diagnosis may be reached by exclusion. Transillumination may, with the test by position of the head, help to exclude maxillary sinus disease. The inability to find bare bone with the probe in the ethmoid region may tend to exclude suppuration of these cells, the position of the pus anteriorly and not posteriorly may exclude the sphenoid trouble, but none of these tests is entirely satisfactory, and on the other hand any or all of these accessory sinuses may be simultaneously involved. When there is pus in the maxillary sinus it is always impossible to say whether it originated there or whether it came originally from the frontal sinus. If, however, the antrum is packed full of gauze the

pus if it originates in the frontal sinus should then appear in the nose. This same thing may happen in ethmoid disease. After antrum disease has been excluded and we are unable to find bare bone in the ethmoid region we may with some degree of confidence look for pus in the frontal sinus.

Treatment.

Lichtwitz and Schaeffer have recommended the opening of the sinus through the nose by the natural passage. Attempts at this have in my hands usually failed. The anatomical arrangement is such that this would be a perfectly feasible procedure, were it possible to illuminate the region and be sure that the probe was engaged in the right orifice. The danger of breaking into the orbit or wounding the ethmoidal region and still more the probability of ultimate failure of this procedure render the external operation the preferable one. A curved incision is made, starting at a point opposite the middle of the eyebrow and two or three lines beneath it, and is carried inwards and downwards to a point opposite and external to the lachrymal tubercle. The skin and periosteum are raised, care being taken not to destroy the pulley of the internal oblique muscle. The under surface of the frontal sinus being thus exposed, enough of the bone is removed with chisel and forceps to allow of free access to the cavity, which is thoroughly explored and curetted if necessary. Or the incision may be made over the frontal prominences above the eyebrow and a little to one side of the median line. The resulting scar is, however, more noticeable. But in either case care must be taken not to destroy the curved edge of the orbit. A strong probe is passed downwards into the nose so that a large drainage tube may keep the passage freely open. The external wound, having been made as aseptic as possible, is then closed with fine sutures and painted with iodoform collodion. A compress is used externally and an attempt is made to get primary union. This is the method advocated by Luc. The frontal sinus in the mean while is flushed out thoroughly several times daily through the tube in the nose, with warm sterile salt solution or warm boric-acid solution. Care should be taken that the drainage tube does not slip into the nasal chambers before it is desirable to remove it altogether. The closure of the external wound at the time of operation, if primary union is obtained, obviates a deforming scar, and if possible this method should be tried in suitable cases. It may not succeed for several reasons:

1st. The drainage tube cannot be kept pervious enough to drain the cavity thoroughly;

2d. It may not be possible to obtain primary union;

3d. If both of these difficulties are overcome the suppuration may still continue after the withdrawal of the tube.

It will then be necessary to reopen the cavity and after exploring it satisfactorily to pack it for several weeks with iodoform gauze, keeping the passage into the nose freely opened by a drainage tube or a piece of gauze. At the end of that time an attempt may again be made to close the external opening and cleanse through the nose.

Mucocele.

Accumulations of serum and of mucus in the frontal sinus have been frequently reported. Their pathology is the same as in similar affections of the antrum. There may be a continuous or an intermittent discharge of clear fluid or of mucus from the nose, or there may be no such discharge, but distention of the inferior walls of the sinus. This will depend on the patency of the fronto-nasal opening.

Without the definite localization of pain and tenderness or of external swelling the differential diagnosis of collections of clear fluid in the various sinuses is impossible. A watery discharge from the nose with general or frontal headaches and heaviness are not uncommon clinical phenomena. Perforation of the inner wall of the maxillary sinus with a trocar is advisable in these cases for diagnostic purpose. Occasionally the operator may penetrate into the frontal sinus through the fronto-nasal duct from the nose. This should always be tried. An external opening of the frontal sinus for diagnostic purposes is not a procedure that recommends itself to the conservative rhinologist and will not usually be allowed by the patient. There are cases, however, where the symptoms are so urgent and yet so inconclusive that the procedure may be wisely undertaken. Where mucous or serous collections exist removal of the cause—nasal obstruction—as spoken of above, by the establishment of a permanent free opening into the nose, will usually cure the case.

Œdematous polypi are also occasionally found in the frontal sinus. Their pathology and etiology are practically the same as in the antrum and in the nasal cavities. They are inflammatory products and should be removed by curetting, but they will return if the suppuration is not subdued.

Tumors.

Osteomata, cysts, and fibromata are the benign growths that have been reported as occurring in the frontal sinuses. The osteomata rarely cause any trouble during life. Pain and distention of the

cavity, with absorption of bone, have been reported in the case of cysts and fibromata. They are exceedingly rare. Sarcomata and carcinomata are also very rare, and when they do occur usually betray their presence only after they have advanced so far as to make operation impossible.

Foreign Bodies.

Foreign bodies in the frontal sinuses from traumatism have been reported. A full description of maggots or of the screw-worm which finds its way into the frontal sinuses from the nose may be found in Morell Mackenzie's work (edition of 1884, Vol. II., p. 439) and in Bryan's article in Burnett's "System of Diseases of the Ear, Nose, and Throat." It is said to be a not uncommon affection in tropical countries among herdsmen.

The Ethmoidal Sinus.

Schech and Bryan both give the order of frequency of suppuration in the accessory nasal cavities as maxillary, ethmoidal, frontal, sphenoid. Bosworth has stated, however, that in his experience by far the most common is purulent ethmoiditis. This is certainly my own experience, but I must hasten to say that I believe that the ethmoid cells proper in a great many of these cases are not invaded, the disease existing in the superficial recesses of the bone only.

It has been shown that it is impossible to separate the etiology and pathology of frontal and maxillary sinus disease, in many cases, from disease of the nasal chambers proper. This is still more true of the ethmoidal sinuses. The ethmoid really enters much more into the formation of the nasal chambers than any other bone and its labyrinthine passages communicate more directly with the nasal chambers than any of the other sinuses. By its position it helps to close in the opening and form more or less of the limiting walls of all the other sinuses.

Acute Catarrhal Inflammation.

We know little or nothing of the acute catarrhal affections of the ethmoidal cells. It is probable that they occur frequently in severe attacks of coryza, but their pathology and their differential symptoms are at present entirely unknown. The same may be said of the acute suppurative processes, and we are even unfamiliar with any chronic suppuration of the mucous membrane of the ethmoidal sinuses without the presence of bare or carious bone. Now from analogy we have every reason to believe that all these conditions must exist. They yet remain to be described.

Purulent Inflammation.

When we speak of ethmoidal suppuration we always involuntarily think of a bone disease. The exceedingly thin and delicate membrane which lines the walls of the ethmoidal cells covers bone structure quite as thin and quite as delicate. The thin plates of bone, too, are lined on both sides by the mucous membrane and exposed on both sides to the air, although sheltered by their position in the middle of the head.

A chronic inflammation of the mucous membrane of the lower regions of the nose may exist for years, even throughout the lifetime of the patient, without the involvement of the bone, protected as the inferior turbinated and the septum are by a tough and more or less thick periosteum and vascular mucous membrane.

Atrophy of the inferior turbinated bone we are familiar with in atrophic rhinitis due to the destruction of its blood supply by the process in the erectile tissue, but we are much more familiar with the hyperplasia of bone and the formation of bony cysts in the region of the middle turbinate than with its atrophy.

The hypertrophic stage which, at least in adults, usually precedes the atrophy of the mucous membrane is accompanied by an increase in the volume of the middle turbinated bone.

In hypertrophic rhinitis it is the soft erectile mucous membrane of the inferior turbinate which causes obstruction of the inferior meatus, but it is bony thickening which usually causes the chief obstruction in the middle meatus.

Noting these anatomical and clinical phenomena we need not be surprised at the prompt involvement of the bone in the suppurative affections of the mucous membrane of the ethmoidal region.

A simple hypertrophic rhinitis extending upwards involves the mucous membrane of the ethmoid in its processes and having previously obstructed the drainage channels below, it renders the conditions favorable for the retention of secretions and the formation of pus in the recesses of the mucous membrane of that region.

Simultaneously with this or in consequence of it, œdema begins in this mucous membrane, and we have the appearance clinically which we call polypoid degeneration and growths which we call nasal polypi. These tend still more to block up the secretions. The pressure of this œdematous tissue mingled with purulent matter may even cause a bulging of the bridge of the nose and exophthalmos. The removal of the polypi with a snare gives vent frequently to a gush of pus which was pent up behind them. Examined carefully, pieces of bone may be found in the mass. These pieces of bone apparently

are embedded in the polypoid tissue. As a matter of fact we have in this condition an œdematous infiltration of the mucous membrane covering both sides of a plate of bone which *in situ* was the dividing wall between two ethmoidal cells or between one and the nasal chambers. This is the phenomenon which led Woakes astray in his "necrosing ethmoiditis" and is the explanation, I believe, of the bone reported as having been found in nasal polypi by Zarniko and others.

Now it naturally follows that pus flowing from a maxillary or frontal sinus over the ethmoidal region may in a like manner set up this œdematous inflammatory condition with the formation of pus, and denudation of bone, with its subsequent crumbling and molecular disintegration. There is no necrosis or death of bone from lack of nutrition. There is clinically no odor of it, but there is the odor of decomposing pus and mucus. The middle turbinated bone is an ethmoidal structure and we frequently get crumbling denuded bone and œdematous mucous membrane, in the folds of which secretions may collect and become purulent; but until the ethmoidal cells become involved, until the process has spread upwards and inwards to this labyrinth, it does not become so grave in prognosis, and is susceptible, I believe, of cure without extraordinary trouble. When, however, the internal walls of these small cavities, many of them with no natural outlet, become the seat of a purulent affection which rapidly denudes and destroys the fragile bone, we have a condition of great gravity because of its proximity to the base of the brain, because of the thinness of the bone which separates the cerebral and nasal cavities at this point, and because the vascular and lymphatic intercommunication is so abundant and so direct.

Fortunately this involvement of the ethmoidal cells is a comparatively rare occurrence. The superficial forms of ethmoid suppuration and bone disease are, however, in my experience very much more common than the suppurations of the maxillary antrum. They are frequently diagnosed as affections of the cell cavities.

Symptoms.

The symptoms of ethmoid suppuration depend so much upon the complicating troubles that it is impossible to draw any distinct separate picture. We may have the symptoms and lesions of hypertrophic or atrophic rhinitis, of nasal polypi, of a deviated and thickened septum, or of a concomitant frontal sinus or antrum disease. There may or may not be pain, and if it does exist it is of such a varying and uncharacteristic description that it helps us little. A purulent nasal discharge from one or both nostrils is complained of for a time extending over months or years.

Diagnosis.

The one point to be depended upon is the distinct feeling of bare and crumbling bone in the ethmoidal region. We may exclude maxillary sinus disease by transillumination or puncture. We cannot exclude frontal sinus disease except by opening the cavity externally. We may exclude sphenoid disease with some degree of certainty if the purulent discharge is seen only anteriorly; on the other hand, we cannot positively incriminate either the sphenoidal sinus or the posterior ethmoidal cells when the pus *is* found posteriorly.

A foreign body high in the nasal cavity covered with lime salts may simulate an ethmoid trouble, but such bodies are rarely lodged so high and are usually movable.

Treatment.

Having excluded suppuration of the antrum and there being no positive evidence of an involvement of the frontal sinus, we may proceed to

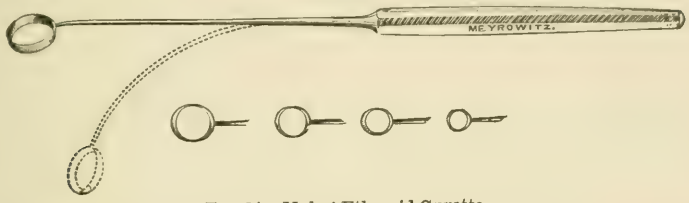


Fig. 24.—Myles' Ethmoid Curette.

treat the ethmoid condition, and the question of further involvement may clear up subsequently. First, of course, all spurs and deviations of the septum on the affected side must be corrected. All polypi and hypertrophies are removed by snare or cautery from the middle meatus or other parts of the nasal chamber. The anterior end of the middle turbinated bone, if enlarged, must be removed. Having gained free access to at least one point of suppuration and bare bone, curettes of size and shape to suit each case are to be used in scraping away granulations and soft bone. For this purpose the delicate instrument

Fig. 25.—Bryan's Ethmoid Curette.

of Dr. Myles may be used (Fig. 24), or Dr. Bryan's ethmoid curette (Fig. 25), a stronger instrument.

Grünwald, in his "Lehre von den Naseneiterungen," which by the way is the best exposition of the general subject which has yet appeared, uses still more formidable instruments. The detritus and pus

should be syringed out with a long flexible silver tube fitted to a large Pravaz syringe.

Using these instruments at the same time as probes, the operator, although working in the dark, by carefully measuring can form a tolerably reliable opinion as to the condition of affairs in the ethmoid labyrinth. Sometimes it is necessary to break through apparently healthy structure in order to reach the seat of disease. Dr. Bosworth recommends that this work can all be done with a burr in a dentist's handle and flexible shaft driven by a hand machine. It will be readily understood that such surgical procedures as this should only be carried out by a thoroughly practised and skilful hand. After intranasal obstructions have been removed and the accessible parts of the ethmoid trouble thoroughly opened up, syringing the parts through the silver tubes spoken of must be done daily or thrice weekly for a considerable length of time (months) until it becomes evident that the seat of trouble has not been and cannot be thoroughly reached and opened up from below.

This is the procedure that I believe to be the best in the uncomplicated cases of anterior ethmoidal disease. When the posterior ethmoidal cells are affected there is usually also an involvement of the sphenoidal sinus.

Where there is an accompanying frontal sinus suppuration the same incision as described above for the latter operation may be utilized to remove portions of the lamina papyracea behind the lacrymal duct, taking care not to encroach upon that. All dead bone, granulations, and œdematous tissue should be scraped away and free communication should be made between the two sinuses and with the nasal chambers. This is an extensive and more or less grave operation and the patient must be carefully watched and the wound must be kept open. The cavity should be packed with gauze for a week or two and closed only after all granulations have been scraped away and all dead bone removed. A wide enough opening may frequently be made into the nasal chambers, by removing the anterior end of the middle turbinated bone, to allow of the closure of the external wound at the end of a few days and of treatment of the region from the nasal passages. In uncomplicated cases of ethmoid disease it may be wise to perform this same operation. The posterior ethmoidal and the sphenoidal sinuses may both be opened in this manner, since it is a rare thing to have them affected without the anterior cells.

Mucocele.

The retention of serous or mucous fluid in the ethmoid cells—the so-called *mucocele*—is a rare occurrence. Bryan says there are only

seven cases on record. They may push into the orbit, causing exophthalmos, or present as a swelling at the inner canthus.

Tumors.

The occurrence of mucous polypus or of polypoid degeneration we have already considered. *Osteomata* of the ethmoid presenting as hard tumors at the inner canthus of the orbit are occasionally seen.

Primary malignant growths of the ethmoidal cells are not unknown, but it is more common for such growths, sarcomata or carcinomata, to start in the naso-pharynx or on the septum and spread to the ethmoidal structures. In either case cerebral symptoms develop and death rapidly ensues. Any extensive involvement of the ethmoidal cells by malignant disease renders the case inoperable.

Syphilis.

Syphilis in the tertiary stage, in the form of gumma, may involve the ethmoidal region and indeed be the primary cause of ethmoidal suppuration. Iodide of potassium will eliminate the specific element, but the suppuration must then be treated on the lines already laid down. Gummatous infiltration of the ethmoidal cells, following the rule, I believe, of all ethmoid disease, does not occur nearly so frequently as does a gummatous deposit in the ethmoidal turbinated bone. When the ethmoidal cells are affected cerebral symptoms soon appear, because tertiary syphilis is a disease which, especially in this region, quickly attacks bone. Indeed, the smell of dead bone in the nose is by far the most reliable sign we have of the presence of syphilis. The odor of dead bone, however, may be absent for some time or be only intermittently present. Sooner or later, however, it is sure to occur.

Apparently tertiary syphilis attacks the other accessory sinuses only very rarely, but in bone disease of the ethmoid it must always be thought of, and frequently the iodide of potassium must be given to clear up the diagnosis. When the diagnosis is once made, however, antisyphilitic treatment, including especially the iodide, must be vigorously pushed, as destructive bone disease in this region is a grave affection.

The Sphenoidal Sinus.

A certain number of undoubted cases of purulent sphenoidal sinus disease are reported from time to time, but they are usually complications of similar ethmoidal affections. The accuracy of the diagnosis in many of the cases may well be questioned; it is always obscure and even the probe entering a suppurating cavity in that region does not always give satisfactory evidence.

The constant presence of pus in the naso-pharynx trickling down from the top of the choana along the septum or the lateral wall, and headaches referred to the occiput are the distinguishing symptoms of suppurative inflammation of this sinus. Pus coming from a pharyngeal bursa—the so-called Thornwaldt's disease—may be mistaken for evidence of suppuration of the sphenoidal sinus or vice versa.

Retention of pus or serum and mucus may cause a distention of the inferior wall into the choanæ, or pushing forward at the back of the orbit may produce exophthalmos. The proximity of the optic nerve is the explanation of the sudden blindness or of atrophy of the optic disc sometimes seen in these cases. This is especially true of tumors of the sphenoid.

Usually, however, the diagnosis of sphenoidal sinus suppuration is made only during the course of treatment of suppuration in the other sinuses. After the obstructions are cleared from the middle meatus the freer access thus gained to the deeper parts of the nose may permit of a positive diagnosis which was at first only surmised.

Bosworth and others regard sphenoidal sinus disease as of rare occurrence, but of very grave prognosis when it does occur. Anatomically this should not be so, because the contents of the cranium are usually very much more securely protected by the thickness of bone than in the case of the ethmoid. We may surmise that the diagnosis is more commonly reached in the severe cases. There have been but few autopsies to settle these points.

The nasal chambers having been freed from obstructions anteriorly, a strong curette or a long trephine driven by a dental engine may be used to perforate the anterior wall of the sphenoidal sinus, which may be found just behind and a little above the posterior end of the middle turbinated bone. This opening may be enlarged by forceps working on the principle of Schroetter's laryngeal forceps. The sinus may then be treated by syringing and packing as indicated in the case of the other accessory sinuses.

Where the orbital incision has been made for the ethmoidal suppuration, access has also been gained by this route to the sphenoidal cells, but their deep situation renders this procedure less satisfactory than in the case of the other sinuses.

Naso-pharyngeal fibromata, sarcomata, and carcinomata often invade the sphenoidal sinuses in an early stage of their course and doubtless occasionally take their origin in the cavity.

Tuberculosis.

Tuberculosis of the accessory sinuses is rarely noted and when it occurs is usually merely an incident in the general affection.

DISEASES

OF THE

NASO-PHARYNX AND PHARYNX.

BY

E. J. MOURE,

BORDEAUX.

DISEASES OF THE NASO-PHARYNX AND PHARYNX.

THE NASO-PHARYNX.

Topographical Anatomy.

We do not propose to dwell long upon the anatomy of this region, but simply to recall the parts which compose it in order that we may the better appreciate the morbid changes in them that will be the subject of this study.

The naso-pharynx is the region lying between the pharynx below and the nasal fossæ above and in front. This cavity is bounded anteriorly by the posterior portion of the septum narium and the orifices of the nasal fossæ (choanal openings) above and by the velum palati below. The posterior wall is formed by the anterior arch of the atlas and the body of the axis, while the vault or upper boundary of the cavity corresponds to the basilar apophysis of the occipital bone. This surface runs very obliquely from above downwards and from before backwards, forming with the posterior wall an angle of about 130° which increases as the head is inclined backwards.

The cavity terminates below at the boundary between the naso-pharynx and the pharynx properly so called. The lateral walls are formed, from behind forward, by a more or less deep groove (Rosenmüller's fossa) and by a rather prominent elevation bounding this groove anteriorly, which is simply the posterior lip of the orifice of the Eustachian tube. This orifice looks downwards and a little inwards, its mean vertical diameter being about half a centimetre. The anterior lip, less prominent than the posterior, is hardly to be distinguished above the level of the lateral wall of the pharynx. This wall is in relation with the great vessels and with the nerves which accompany them (the hypoglossal, glosso-pharyngeal, and sympathetic).

The following description of the mucous membrane lining the naso-pharynx is borrowed from my treatise on "Diseases of the Nose" (second edition, Paris, 1893): The mucous lining is continu-

ous posteriorly with that of the pharynx and anteriorly with that of the palate and nasal cavities. It differs, however, from the latter by its more uniform thickness and by the presence in it of closed follicles situated chiefly in the neighborhood of the Eustachian tube, in the fossette of Rosenmüller, and especially on the upper wall or vault of the pharynx. In the latter situation the follicles are sometimes so numerous as to have received the name of the pharyngeal tonsil (Robin, Kölliker) or Luschka's gland, so called after the author who has given an excellent description of it.

Following Mégévand we shall consider the pharyngeal tonsil as a special organ having a pathology of its own. In its normal condition this gland appears under the form of nipple-shaped projections, separated by antero-posterior grooves symmetrically arranged on each side of the median line; these divide the tonsil into several lobes, three according to Trautman, four according to Robin, or a variable number according to our own view. The more or less deep grooves give to the organ a very fair resemblance to the cerebellum (Mégévand). The pharyngeal tonsil has a clear pink color, sometimes, however, inclined to yellowish. It is supplied by the vessels which will be mentioned later as existing in this region. We shall see in the course of this article that hypertrophy of this tissue may entail grave consequences, which have been recognized and described only within the past few years.

The epithelium in the neighborhood of the Eustachian tubes is ciliated, but is of the pavement variety in all the other portions of this cavity.

At the posterior part of the pharyngeal tonsil is found a depression which was first described by F. J. C. Mayer, later by Luschka, Ganghofner, Tortual, and others under the name of the pharyngeal bursa. The orifice of this bursa, sometimes rounded but more often slightly elongated transversely, has a diameter of from 2 to 5 mm. The body of the bursa is dilated, and terminates sharply at the periosteum of the basilar apophysis, which often presents a slight depression at this point; its depth is 8 or 10 mm. in the adult, 4 or 5 mm. in the child. Its wall is formed by a cellular membrane having subepithelial elastic fibres. Outside of this is a layer of connective tissue containing here and there a few follicles, which are of very small size in the child.

It is to be added, however, that the existence of this bursa has been doubted, and the recent researches of Schwabach, Gallé, Range, and myself tend to prove that this recess is the remains of a more or less atrophied adenoid vegetation, of which the median groove has persisted.

Pharyngoscopy and Posterior Rhinoscopy.

It would seem at first thought as though the examination of the pharyngeal cavity might be made most naturally by having the subject open his mouth, while facing the sunlight or a good artificial light, the examiner simply depressing his tongue. However, if we remember that the pharynx is composed of two distinct parts, one buccal, the other nasal, we can readily understand that, while the inspection of the first part may be comparatively easily made in this simple manner, the examination of the nasal pharynx calls for the employment of a somewhat more complicated method.

We need not dwell here upon the description of the means of illuminating the pharyngeal cavity during an examination, the apparatus employed for this purpose being described elsewhere. Neither is it necessary to describe in detail the mode of examination of the buccal pharynx. Certain patients, notably singers, submit to this without the aid even of a tongue depressor, themselves flattening the tongue at will so as to expose to full view the posterior wall of the pharynx, but it is not every one who can do this, and some persons, immediately they open their mouth, arch up the tongue involuntarily. In many of these any attempt on the part of the physician to depress the tongue instrumentally causes nausea or vomiting, and some are so sensitive that even the simple opening of the mouth for inspection causes nausea, that may go on to retching and vomiting if the attempt is persisted in. One of my patients was unable to have anything done to his mouth, teeth, or gums without experiencing nausea that compelled the cessation of all treatment. Such cases are, of course, exceptional and are not to be considered at all in formulating rules for the conduct of these examinations. In a general way, in making an examination of the buccal pharynx, as soon as the light is properly directed the patient should be requested to open his mouth, and his tongue must then be gently depressed; this may be done and the organ be retained against the floor of the mouth behind the lower teeth by means of the handle of a spoon, a spatula, or, better yet, an ordinary tongue depressor. In order to avoid as far as possible the production of retching, it is well not to put the tongue depressor too far back, but to insert it only as far as may be necessary to flatten the anterior third of the tongue against the floor of the mouth. The patient should now be made to pronounce *ah*, and then to take a deep inspiration in order to expose to view first the upper part of the buccal pharynx through the partial drawing up of the velum palati, and after that the inferior portion of this

cavity, and sometimes the free border of, or even almost the entire epiglottis. If the subject to be examined is especially sensitive, or if the tongue is involuntarily vaulted up, we may try taking the tip between the thumb and finger, guarded by a napkin or handkerchief, and, pulling it from the mouth as in a laryngoscopical examination, have the patient take a deep inspiration, and pronounce *ah* during expiration.

In the case of a child who refuses to open the mouth, the best procedure is to have its head held in the proper position to receive the light from the mirror on the posterior wall of the pharynx, and then, seizing the nostrils between the thumb and the index finger of the left hand, to await the favorable moment afforded by inspiration to introduce the spatula between the partly opened jaws. Once this has been accomplished we may easily force the child to open its mouth wide by inducing retching. The opportunity afforded by this wide opening should then be utilized by the introduction of a wedge of some sort, a cork, a piece of wood covered with cloth, or a gag, in order to keep the jaws separated and to prevent injury from the child's teeth when the finger is introduced.

Sachs recommends tickling the fauces, in order to produce these reflexes, by means of something passed between the teeth and the cheek and turned into the fauces through the space existing in the child behind the molars. This is very easily done.

The first object which strikes the eye in the examination is the uvula, which, when healthy, is of a pale pink color like that of the palpebral conjunctiva. The free border of the velum palati is seen stretching in the form of a crescent, from the lateral border and base of the uvula on each side, to become continuous laterally with the pillars of the fauces, between which lie the buccal tonsils. In the space comprised between the pillars and the velum, but a little further back, we see starting from a sort of ledge a vertical surface, covered with mucous membrane that is of a somewhat deeper pink color, even in health, than the neighboring parts of which we have just spoken. This smooth, rather brilliant wall, sown here and there with little elevations denoting the site of the mucous glands, is the buccal plate of the pharynx.

Examination of the Naso-Pharyngeal Cavity.—After having inspected this part of the posterior wall of the pharynx it will be necessary to complete the examination by means of the pharyngeal mirror which enables us to see the opening of the posterior nasal fossæ. The exploration of these parts is, however, sometimes accompanied by such difficulties as to impede or even prevent us from attaining our ends. In children especially, the fright that the sight of the instru-

ments occasions is very often sufficient to prevent the employment of this method of investigation. Again, in adults, the position of the soft palate, almost glued as it were to the pharynx, or the considerable and abnormal mobility of the posterior wall of the pharynx, offers an obstacle to the introduction of the rhinoscopic mirror. These impediments may now be overcome by means of cocaine applications, which cause complete anæsthesia of all the parts and consequently abolish the reflexes interfering with the examination.

To obviate the first impediment which we have mentioned (the pressing of the soft palate against the posterior wall of the pharynx), some authors have proposed to make use of a uvula elevator, attached to the mirror (Duplay) or held in one hand, while the tongue depressor and the mirror, united in one piece, are held in the other (Czermak and Voltolini), or else forming one instrument with the tongue depressor. The usual palatine hooks (Voltolini, Schrötter),

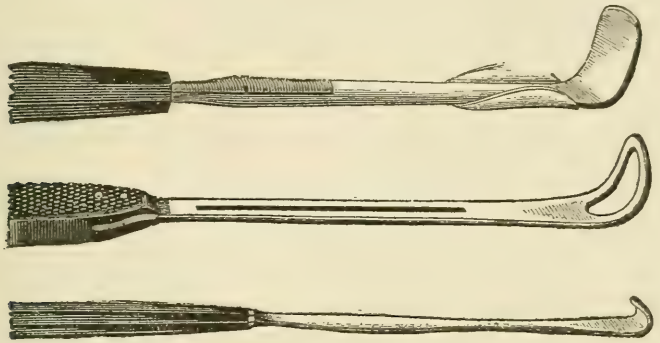


Fig. 26.—Palate Hooks; one-half the actual size.

plain or fenestrated (Fränkel), are, except the latter, made with two little wings forming two lateral gutters, in one of which rests the uvula raised during the examination.

We must admit, however, notwithstanding the introduction of cocaine into medical practice, that these various instruments are not as a rule altogether practical. But it is very different with the elevator of Krause or that of Moritz Schmidt, of which the accompanying illustration (Fig. 27) will give a clear idea and render a detailed description unnecessary. Before using these instruments it is important to anæsthetize the pharynx and naso-pharynx by the insufflation of a mixture of sugar and cocaine in equal parts—a small pinch being ordinarily sufficient to produce the desired result. The velum being raised, a large orifice is exposed to view through which it is very easy to pass the rhinoscopic mirror.

In making an examination of the naso-pharynx, the observer will

place the patient facing him with the head slightly projecting forwards. The light should be the same as that used in laryngoscopic examinations, projected by the ordinary head mirror. The rhino-

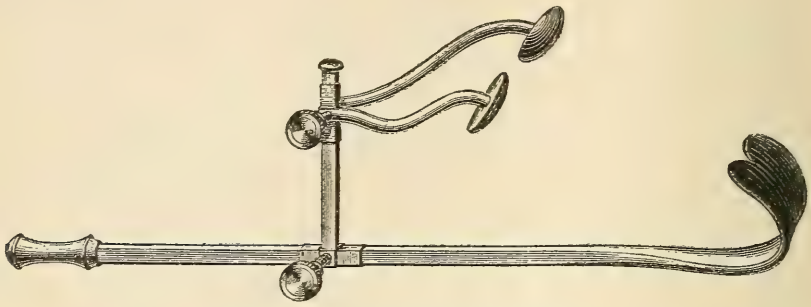


Fig. 27.—Moritz Schmidt's Palate Elevator.

scopic mirror is similar to that used for the inspection of the larynx, but much smaller, and of a round or oval shape, preferably the latter, since such a mirror takes up much less space in an antero-posterior sense. Some authors recommend the use of a mirror fixed at a right angle with the handle, but it seems to me that an inclination of 135° to 145° is much better for those who are not very familiar with this method of examination.

After the patient has been made to open his mouth wide and breathe quietly through the nose, the light is thrown into the throat. Then, taking an ordinary tongue depressor in the left hand, the examiner depresses with some force the patient's tongue against the floor of the mouth, while with the right hand he introduces the mirror in the direction of the posterior wall of the pharynx, the reflecting surface being directed upwards. In some cases the tongue is so well depressed and flattened by the patient's own efforts that the pressure with the handle of the mirror alone will suffice to keep it there.

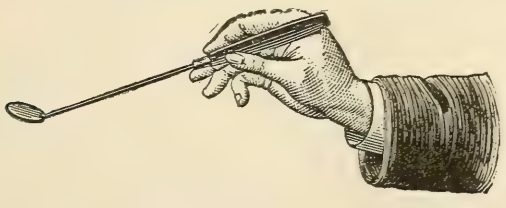


Fig. 28.—Position of the Hand and of the Mirror during a Posterior Rhinoscopic Examination.

A precaution which it is always useful to observe upon reaching the level of the soft palate is to turn the mirror vertically so that it will occupy the least possible space and may pass between the uvula and the anterior pillar without touching these parts and consequently without producing any reflexes which will render the examination impossible.

Once the mirror is in place, if nasal respiration does not suffice to increase to its maximum the space comprised between the velum palati and the wall of the pharynx the patient must be asked to pronounce some nasal sound, such as the French *bon*, *ban*, or *bein*. In consequence of the small size of the mirror employed it is almost impossible to perceive at one time the entire extent of the posterior nasal fossæ, so that it is indispensable, in order to obtain a complete idea of the condition present, to incline the mirror successively in different directions. It is by joining together such a series of images that we are able to obtain a complete view of the posterior nasal cavity. When the velum palati is widely separated from the posterior wall of the pharynx we may make use of the ordinary laryngoscopic mirror and thus obtain a view of the entire cavity.

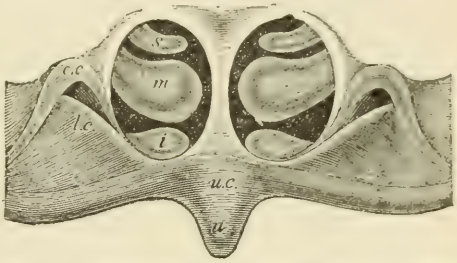


Fig. 29.—Rhinoscopic Image of the Posterior Nares.

When the mirror is almost horizontal we obtain a view of the superior wall or vault of the pharynx, which is irregular and presents here and there little oval or rounded depressions, or simple grooves of greater or lesser extent, indicating the seat of the pharyngeal tonsil. In the normal state these irregularities are very slightly marked, especially in the adult, and one must look carefully in order to distinguish them.

A slight inclination of the mirror to the right or left gives us a view of the lateral parts, where we see Rosenmüller's fossa, bounded anteriorly by the posterior lip of the pharyngeal orifice of the Eustachian tube, the latter showing as a yellowish-white spot in the mucous membrane. If we turn the reflecting surface of the mirror upwards and forwards we see in the median line, anterior to the superior wall, the septum narium, through the thin transparent mucous membrane of which we perceive the yellowish color of the underlying bone. This septum, usually inclined a little to the right or to the left, is thicker above than below.

On each side are seen the posterior openings of the nasal fossæ (choanæ), in the upper part of which near the septum we see a slight projection, which is the superior turbinated body. Below we notice a more or less pronounced shadow which represents the superior meatus, and a little below this appear the middle turbinated bodies under the form of two oblong tumors, glistening, of a dull-red color inclined to grayish.

By inclining the mirror still further forwards we discover the mid-

dle meatus, then the inferior turbinated bodies, of which as a rule we can distinguish only the upper portion, having the form of two rounded projections of a pale pink color, a little more distant from the septum than the middle turbinated bodies. The lower portion of the third turbinated body and the inferior meatus are generally invisible, the velum palati forming at the root of the uvula an arch of a grayish-pink color which hides these parts. When, however, the pharyngeal cavity is wide, we may, by introducing the mirror to the extreme posterior portion, obtain a view of almost all the lower turbinated body, as well as a good part of the floor of the nostril. If we have to do with patients who are difficult to manage, whether because they refuse to open the mouth, as in the case of children, or because they are unable to depress the tongue, we may try direct examination of the naso-pharyngeal cavity through the nostrils, by means of Zaufal's speculum, which gives especially satisfactory results in the case of patients having wide nostrils as a result either of a natural conformation or of a pathological atrophy (atrophic coryza). But in these cases the examination is evidently less satisfactory and ought always to be followed by digital exploration.

Digital Exploration.—Independently of the notions furnished by the sense of sight, touch may also give useful information, especially in the case of children, whom it is impossible to examine satisfactorily with the rhinoscopic mirror. The finger curved in the shape of a hook and carried rapidly behind the soft palate seems to us to be more useful than sounds or other instruments of the kind, at least as an aid to diagnosis. It is also useful to make a practice of digital exploration of the posterior nares in order to learn how to manipulate the finger in this cavity, and especially in order to familiarize one's self with the projections which exist normally in those irregular passages, so as not to expose oneself to grave errors of diagnosis. Finally, whenever possible, it will always be advantageous to combine direct inspection and palpation, for by means of the latter we may substantiate the results of inspection.

General Therapeutics of the Pharynx and Naso-Pharynx.

In the treatment of diseases of the pharynx and naso-pharynx we may employ: (1) Liquids; (2) vapors; (3) air impregnated with medicinal substances; (4) powders; (5) liquid or solid caustics or the thermo-cautery.

1. *Liquids.*—These are applied to the pharynx in the form of gargles or local douches, which consist in the introduction into the mouth of a hot, lukewarm, or cold liquid, according to the special indications,

and bringing it in contact as fully as possible with the parts constituting this region. In gargling, the best way of making the liquid bathe all parts of the pharynx thoroughly is that described by Moura-Bourouillou, which consists in making the patient pronounce the syllables "*glou-glou*" while holding his head inclined far backwards. We must remember that in many cases the tongue resting against the palatine vault prevents the fluid from bathing the tonsils and still more the pharynx, as is intended. At other times, on the contrary, not only may the fluid reach the pharynx but it may even enter the naso-pharynx and be returned by the nostrils. This sort of retronasal lavage, which was recommended by Guimier de Montpellier, is obtained by carrying the head rapidly from behind forwards and, at the same time, breathing out through the nostrils in much the same way that smokers blow tobacco smoke through the nose. This procedure has the grave disadvantage of causing a liquid which has been fouled by its passage through the mouth to bathe the naso-pharynx. We prefer, in order to obtain the same results, another method which we shall describe below.

Lavage.—In cases in which we desire to obtain a more energetic effect and a true washing out of the pharynx, we make use of the douche, or irrigation, which is practised in different cases with the ordinary siphon, the irrigator, or a syringe. We need not mention here the different remedies employed in these topical applications since they vary with almost every disease; in general they are of an antiseptic nature.

In order to make this lavage, which is especially employed in children suffering from severe sore throats who do not know how to gargle, it is well to have the head of the patient held forwards, over a basin. We ought, whenever possible, to make the injection during expiration (which we bring about by making the patient pronounce the sound "ah"), interrupting from time to time the flow of the liquid. This method is very easy when we have to do with affections of the pharynx properly speaking, but it is altogether insufficient in certain diseases of the naso-pharynx. In these cases we prefer to replace the retronasal gargle, of which we have spoken above, by the local douche made with a suitable cannula, the employment of which we have systematized in the case of adults. But the retronasal douche no more than the gargle is suitable for children under ten years of age; though it is well to add that we have less frequent occasion to employ this mode of local treatment in children than in adults.

I also do not believe that lavage from behind the velum palati can be replaced by Weber's douche, the liquid passed through the

anterior nasal fossæ failing to reach the greater part of the naso-pharynx. Moreover, the retronasal injection is usually well borne by the patients who, in the majority of cases, readily acquire the facility of practising it themselves. In order to facilitate this manœuvre I usually employ a retronasal cannula pierced with four holes only, attached to an ordinary enema syringe; in this way the liquid is injected intermittently, the patient being able to prolong or shorten the interruption at will; on the other hand, the cannula, being perforated by four holes only, does not give exit to a large quantity of fluid, inundating the pharynx.

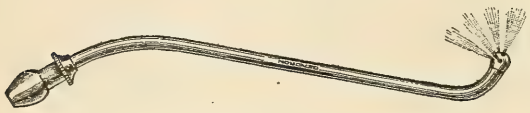


Fig. 30.—Cannula for the Retronasal Douche.

Of late years nasal and retronasal douches have been accused of causing the penetration of septic fluids into the Eustachian tubes, but the fear of this accident has certainly been greatly exaggerated. The accident is more apt to occur when the nasal passages are obstructed so that the injected fluid does not find a ready exit, that is to say, when the injections are really contraindicated. The physician must of course know how to prescribe this method of treatment, and especially be able to recognize the cases in which it ought not to be advised. If we employ these douches with discretion we shall find that, far from being injurious, they are capable of rendering excellent service in the treatment of diseases of the naso-pharynx.

Sprays.—We may also carry fluids into these regions by means of apparatus called spray producers, all constructed on the same general principle. We need not describe here the different instruments employed for this object, for they vary in shape with each country. We need only remind the reader that the different spray producers

Fig. 31.—Retronasal Atomizer.

may be brought under certain special types, to wit: (1) Those in which the fluid is propelled by means of a single or double bulb (Richardson); (2) steam atomizers in which a medicated liquid is

drawn up and projected by means of steam, giving a warm spray; (3) another form, employed specially in thermal stations, in which the water, projected under a certain determined force, is atomized in striking against a palette or, carried through a rather fine tube, it is passed through a sieve with finer or coarser meshes, which throws it into a spray varying according to the pressure and to the style of apparatus employed. The fluid thus thrown constitutes a pharyngeal douche rather than a spray.

It is hardly necessary to add that according to the shape of the tip of the spray apparatus fluid is directed into the throat or into the naso-pharynx.

Direct Application.—It is not uncommon, when we wish to obtain a more energetic action than follows the use of gargles or of the douche, to apply a remedy by means of a brush, sponge, or pledget of cotton attached to a metallic shank firmly set in a solid handle. A piece of cloth wrapped around the end of a stick is very commonly employed for this purpose, but we much prefer absorbent cotton, which is readily impregnated with the medicated liquid and may be renewed each time. It is needless to add that the shank should have a curve adapted to the special region to which the application is to be made. If the liquid employed is of a caustic nature, we must take care to press out all surplus from the brush, sponge, or pledget of cotton before introducing it into the pharynx.

2. *Vapors.*—In some acute inflammations of the pharynx it may be useful to apply warm vapors to the mucous membrane. This is done by boiling in a suitable receptacle the medicinal agents prescribed and conducting the steam from this vessel to the part desired by means of a hood of cardboard, metal, glass, or porcelain, or by simply instructing the patient to hold his head over the vessel, the steam being retained by a towel over the head. The steam given off is then inhaled by the patient, and it also acts as a vapor bath for the entire head. The same result is obtained at certain bathing establishments by having the patients remain in a hall containing the hot vapor of some mineral water. The steam atomizer, of which we have spoken above, is another very common means of applying vapors to the pharynx or naso-pharynx.

3. *Air Impregnated with Volatile Principles.*—In other cases patients are made to breathe, not vapors, but simply medicated air. Although this is of more common employment in affections of the nasal fossæ, properly speaking, or of the larynx and air tubes, it is occasionally used also in certain diseases of the throat or posterior nares.

These topical applications are employed chiefly in thermal stations where there are gases such as sulphureted hydrogen, carbonic acid,

etc., given off from the mineral waters. It is necessary also that the temperature of the water should be sufficient to volatilize the gaseous principles and saturate the air to be inhaled. Some of the spas in France are admirably adapted to this purpose.

In other cases certain volatile substances such as menthol, thymol, balsam of Peru, eucalyptol, etc., are employed in this manner. These various medicaments are usually dissolved in alcohol so that their dosage may be properly graduated, and are then dropped into hot water so as to impregnate the air surrounding them. We have employed for several years the apparatus here illustrated, which by reason of the mask with which it is provided permits of the passage of the impregnated air through the nose into the naso-pharynx or directly into the pharynx itself.

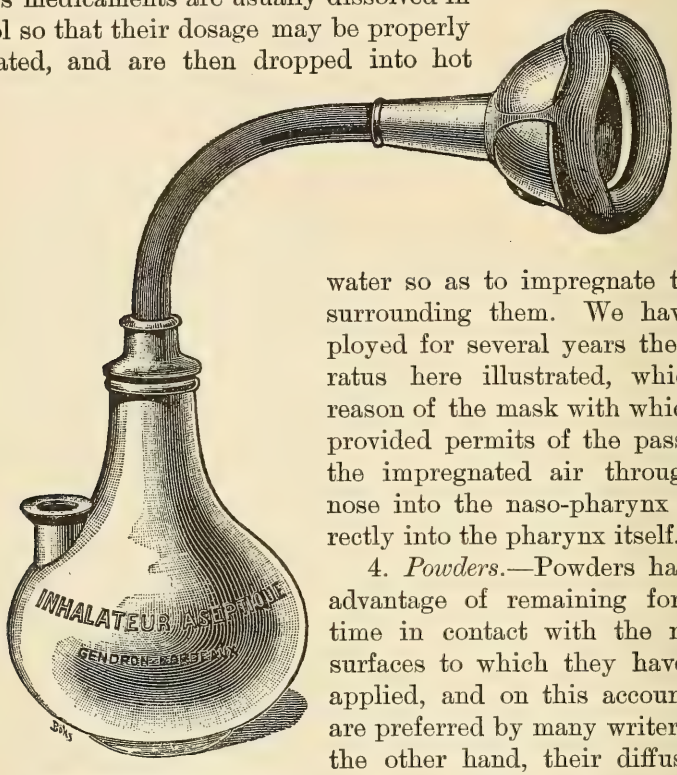


FIG. 32.—Antiseptic Inhaler.

4. *Powders.*—Powders have the advantage of remaining for some time in contact with the mucous surfaces to which they have been applied, and on this account they are preferred by many writers. On the other hand, their diffusibility and the difficulty of limiting their action render their employment inadvisable in the case of local inflammations. The active principle is usually mixed with an inert soluble powder such as borax, sugar, or the like, which serves as a vehicle, and the powder is introduced into the throat or posterior nares by means of insufflators, which vary in form in each country. The insufflator of Kabienski is most employed in Germany, while in France and in America we often make use of a simple rubber bulb, blowing the air through a tube, which is straight or bent according as it is desired to project the powder in one or another direction. It is easy to throw pulverized medicaments into the naso-pharynx through the anterior nares by introducing Zaufal's funnel speculum into the nasal cavity.

5. *Caustics.*—By means of the different applications which we have just considered we are able to cleanse or to medicate the mucous membrane of the pharynx or nostrils as a whole. We will now consider briefly the more energetic applications which act as caustics. These are either liquid or solid caustics or the thermo-cautery.

(*a*) Liquid. The only caustics employed in this form are solutions of nitrate of silver, chloride of zinc, or carbolic acid. Other substances ought not to be employed in liquid form since it is usually very difficult if not impossible to limit their action.

(*b*) Solid. Substances employed in solid form are nitrate of silver (pencil) and chromic acid. These two remedies may be melted on to the tip of a glass rod or a probe. In the case of nitrate of silver the stylet is first heated and the substance melted and applied in this way, but for chromic acid another process is employed. This agent ought to be used after the method of B. Fränkel and Heryng which permits of the localization of its action and prevents pain. A very small piece of the crystallized acid is heated over an ordinary lamp or a gas jet on the extremity of a metallic or glass rod. Its water of crystallization is thus driven off with a slight crackling sound and the substance melts, forming a thick, sticky mass of a dark-brown color which rapidly solidifies when placed on the edge or the tip of a *porte-caustique* according to the desire or needs of the operator. It forms at this point a coating more or less thick, the thinner the better, however, having the dull look and the color of the tip of a safety match. Taking the *porte-caustique* so made, we trace several light lines over the mucous membrane to be cauterized; these lines gradually acquire a yellowish hue and, increasing in width, will gradually coalesce if they were originally made close enough together. After the cauterization has been made the patient should snuff up a little tepid water slightly alkalinized with bicarbonate of soda in order to remove excess of acid in contact with the mucous membrane.

If the caustic had been applied to the tonsils or the pharynx we inject the alkaline solution into the patient's mouth, having him then gargle and instructing him not to swallow any saliva for several minutes. The use of chromic acid in this location has the disadvantage that the substance is toxic in small doses.

(*c*) The Thermo-Cautery. We may employ the thermo-cautery with platinum tips renewed at each application. This instrument, which it is impossible to use in the naso-pharynx, is, on the contrary, very serviceable in certain affections of the buccal pharynx and of the tonsils especially. When used the tips should always be of a dull red and should not be applied for too long a time for fear of burning the neighboring parts through radiation.

The galvano-cautery, on the other hand, is of undoubted utility in the treatment of affections of these regions, because it may be introduced cold, carried to the part which it is desired to cauterize, and there maintained at any desired temperature. It is a useful instrument and one which can be made in any desired form, the platinum tip being made in any shape and adapted to any part. It is of course out of the question to describe here all the different knives, points, and other tips which each operator constructs after his own models. We need also devote no space to the description of the apparatus necessary to produce the electric current. These are in different cases single fluid batteries with bichromate of sodium or of potassium, accumulators of different models, or finally the electric-light current transformed into galvanic heat. This transformation may be obtained in several ways according as the electric-light currents are alternating or continuous. We cannot here occupy ourselves with these technical points, and need only mention the transformator devised a year ago by Dr. Roaldes, of New Orleans, which, in spite of its cost, appears to us to have solved in great measure the problems, at least the economical ones, since it takes up only the current necessary for the purpose desired, whether the heating of a cautery or the lighting of a lamp. We may also employ the city currents by utilizing the resistance of a thread of German silver or an incandescent lamp, giving, according to its intensity, a known number of ampères. This is the procedure which we have employed for several years, and notwithstanding its somewhat high cost it has given more satisfaction than the usual primary or storage batteries.

The cauterization is made, according to the results desired, in the form of punctures or of parallel or radiating lines, the dimensions and form of which will vary according to the needs of each operator. In other cases electricity is employed in the form of the galvanic loop. In the use of this a certain amount of experience is necessary in order not to burn through the thread of the loop before having divided the tissues in which it is buried. The thread should be kept as nearly as possible constantly at a dull-red heat, a result which may be the more easily obtained if we remember to make the interruptions of the current more frequent as the loop is shortened. I first advised this method several years ago, and I have always found it serviceable.

Although accidents are less common after the cauterization of the pharynx than they are after that of the nose, we must nevertheless remember that they may occur. Hemorrhage may occur if the thermo- or electro-cautery is carried to a white heat. The neighboring tissues may be burned in consequence of awkwardness on the part of the operator, indocility of the patient, or radiation from the

instrument. We may also include among the possible accidents cicatricial adhesions, following too deep or too rapid cauterization. These adhesions occur more especially between the posterior pillars of the palate and the pharynx, or between the latter and the vertebral column, following ignipuncture for granulation of the pharyngeal wall.

Electricity.—We may also employ electricity in the form of induced or continuous currents in certain paralytic or vasomotor affections of the pharynx. It is likewise employed rather commonly in order to reduce or destroy certain vascular or inoperable tumors of the different parts by means of its electrolytic action. We shall refer again to this method when we come to treat of various affections in which it may be indicated.

Massage.—Massage is a new therapeutic measure in affections of the naso-pharynx and pharynx which has recently been introduced by Braun, of Trieste, and Laker, of Gratz. It consists in rubbing the mucous surfaces lightly with a stylet covered with cotton moistened in a neutral solution. The stylet ought to be moved rather rapidly in order to produce true vibratory movements in the parts acted upon. The movements should be as regular as they are rapid, and Laker was so skilled in these manipulations that he could produce a regular tracing with the oscillations made by his hands. In order that these rubbing movements may be more light, more rapid, and more regular, the attempt has been made to attach the different stylets to an electric motor, the movements of which could be graduated at will by the interposition of a resistance in the current, the mechanism causing this resistance being regulated by a pedal so as to leave the hands free for the necessary manipulations. This mode of treatment, while not a panacea, may yet be of great service in special cases.

Local Anæsthesia.—In order to obtain local anæsthesia we usually employ a solution of the hydrochlorate of cocaine in ten, fifteen, or twenty-per-cent. solution, according to the individual sensibility of the patient. When the patients are very timid it is well to spray a few drops of the cocaine solution, and then at the expiration of one or two minutes the anæsthesia may be completed by swabbing with a pledget of cotton impregnated with the solution. Instead of spraying a solution on the parts, a powder of cocaine mixed with sugar, as recommended by Schmidt, may be blown into the throat. The degree of insensibility varies with the number of applications and the strength of the solution. The following is a solution that can be recommended.

Hydrochlorate of cocaine, . .	1.00, 1.50, or 2.00
Pure glycerin, ...	2.00
Cherry-laurel water,	
Distilled water, ...	āā 4.00

As far as possible freshly prepared solutions only should be used. Thanks to this anæsthesia we may employ caustics of any strength or the thermo-cautery without causing the patient any pain. We ought to add, however, that the mucous membrane of some patients appears to be refractory to the action of cocaine, and in such case we simply lessen the sensibility of the parts without abolishing it completely, even with the employment of a twenty-per-cent. solution. We shall see that it is especially in patients suffering with certain forms of nervous hyperæsthesia that this resistance to the usual action of cocaine is most marked. It is well to remember also that the remedy, like many others, loses much of its efficacy when applied too often to the same individual. Formerly this agent was employed *larga manu* without the least fear and often, indeed, without occasioning any disturbance; but recently a number of accidents have been reported of such a nature as to cause the practitioner to use the anæsthetic with great caution. Although we ourselves have only very rarely noticed a little pallor of the face or a semi-syncopal state attributable, in part at least, to the apprehension of the patient, we still think that it is wiser to test the susceptibility of the patient first by using weak solutions, one to fifteen for example, using larger doses later if the effect obtained is not sufficiently marked and especially if the employment of the cocaine has been found to produce no ill effects.

Lion has recently recommended as a substitute for cocaine an alcoholic solution of menthol in the strength of thirty to fifty per cent. (Rosenberg) and concentrated solutions of antipyrin, one part to three or in equal parts (Coupard and St. Hilaire), but these agents are far from having the energy of cocaine, to which the preference must unquestionably be given.

We need not speak here of general anæsthetics such as chloroform, ether, or bromide of ethyl, which will find their special indications in individual cases that we shall mention later in the course of this work.

Malformations of the Naso-Pharynx.

Deformities of the naso-pharynx may be congenital or acquired. The latter, which are the more frequent, will be described when we treat of the individual diseases and especially tuberculosis and syphilis of this region, upon which they depend.

Congenital malformations, properly speaking, those which are dependent upon diseases of the fœtus excepted, are in general very rare. The absence of the naso-pharynx has never been reported; but the walls which limit this cavity are susceptible of certain modifications. The anterior wall formed above by the edges of the choanæ

and the posterior edge of the vomer, below by the posterior surface of the velum palati, is the most often affected.

Complete or partial obliteration of the posterior nares by a membranous diaphragm, or more commonly by a bony one covered with mucous membrane, is the most common anomaly. It interferes with respiration through the nose and leads to consequences which are even more serious than those which will be referred to when we come to speak of tumors of the naso-pharynx, the chief of which is the impossibility of the child's obtaining nourishment by the breast. Instances of this obliteration have been reported by Luschka, Voltolini, Betts, Solis-Cohen, Donaldson, Moure, Gougenheim, Beausoleil, and others. They are usually unilateral and formed by a thin plate of bony tissue extending as a sort of curtain between the vomer and the pterygoid apophysis. It is usual to find in the subjects of this malformation a more or less marked narrowing of the opposite choanal orifice. In two cases which I have seen the unclosed fossa would hardly admit the extremity of the tip of the little finger. The treatment consists in the formation of an opening through the obstructing plate with the galvano-cautery, the orifice being afterwards dilated by means of gutta-percha or metallic bougies, when cicatrization is taking place. This operation, which is performed through the nostrils, is often exceedingly difficult. It ought scarcely to be tried in the case of a young child; it is better to wait until adolescence before undertaking it.

Congenital division of the velum palati, which will be considered in the section devoted to the anomalies of this region, is ordinarily accompanied by more or less complete absence of the vomer and by other changes which alter considerably the image obtained in posterior rhinoscopy.

In place of being a plane and vertical surface covered by smooth mucous membrane the posterior wall sometimes bulges out at its upper part by reason of a displacement forwards of the anterior arch of the atlas and axis; and at the same time is observed almost constantly a more marked obliquity of the posterior edge of the vomer, the upper extremity of which is carried backwards in such a way that the upper portion of the wall of the naso-pharynx is narrow and forms as it were the bottom of a deep recess. Into this the finger can hardly be introduced, and if there are adenoid vegetations in this part the ordinary instruments cannot be used for their removal.

Farlow, Sanderson, and others have noted the presence of a large vessel under the mucous membrane of the posterior wall of the naso-pharynx and continued under that of the pharynx. For this reason we ought never to neglect examination of this region before under-

taking any surgical interference with the upper portion of the naso-pharynx.

The posterior wall of the naso-pharynx may also be modified by membranous folds or fibrous bands stretching from the posterior lip of the Eustachian orifice. These folds, sometimes very short, are directed in certain cases transversely, filling up Rosenmüller's fossettes, and are inserted into the median line of the posterior wall of the naso-pharynx, to which they give a concave shape. In other cases they take an oblique direction, still, however, reaching the median line as before, being inserted into the mucous membrane which covers the basilar surface of the occipital bone, thus terminating in the vault of the naso-pharynx. There they form two ridges more or less wide, which constitute a bridge above the fossettes, and by meeting in the median line give to the vault the form of a Gothic arch. This Gothic-arch formation is rather rare, since partial bands and those of small size are more common; nevertheless they are occasionally seen, and I myself have observed them several times.

These different anomalies call for no special treatment.

Acute Naso-Pharyngeal Catarrh.

Definition.—By this term, or, better, by that of acute adenoiditis (Moure), is understood a catarrhal inflammation of the mucous membrane of the naso-pharynx, and more especially of that collection of closed follicles constituting Luschka's tonsil, lying under the vault of the naso-pharynx. The uniformity of the anatomical constitution of the mucous membrane of this region, notwithstanding the unequal distribution of the closed follicles there contained, does not permit of a separation of the study of inflammation of Luschka's tonsil, properly speaking, from that of the upper portion of the pharynx, since clinically these two constitute one and the same disease.

Etiology.

The causes of acute adenoiditis are of two sorts, viz., predisposing and determining. Among the former we must range the lymphatic constitution, scrofula, an especial nervous state, perhaps arthritism, and always youth. The reason why youth predisposes to this condition is that there are frequently present in young subjects adenoid vegetations which keep up a constant state of irritation in the naso-pharynx, making of it a *locus minoris resistentiæ* where external agents act with greater facility.

The determining causes may be certain external agencies or infectious germs, the latter being either already present in the system in

connection with some general disease or introduced from without. Among the number of external agents we may cite in the first place atmospheric changes, either of temperature, of pressure, or of humidity; general or partial refrigeration, especially cold feet; passage from a warm to a cold atmosphere, or the reverse; exposure of the uncovered head to the sun, and exposure to currents of air.

The infectious diseases accompanied by adenoiditis and frequently also by buccal pharyngitis are numerous: among them are scarlatina, measles, typhoid fever, erysipelas and diphtheria; pneumonia, pyelonephritis (Mégévand), and acute tuberculosis must also be mentioned, but they are less common causes. We shall also have occasion to refer to lesions of the naso-pharynx due to infectious disease when we come to mention the anginas of the same name, of which they are only the extension; thus syphilis of the naso-pharynx has been intentionally omitted, its description being reserved for a separate section.

Acute adenoiditis due to infection from digital exploration is far from being rare; it is accompanied, in patients suffering from adenoid vegetations, by an exacerbation of the functional troubles from which they ordinarily suffer, an exacerbation announced by a more or less violent chill and followed in many cases by an elevation of temperature which may reach or even exceed 40° C. (104° F.). Such occurrences emphasize the necessity of rendering the finger aseptic before retronasal exploration is attempted.

Symptoms.

These differ slightly according as the affection is seen in children or adults, the difference being due on the one hand to the absence or small size of adenoid vegetations in the adult, and on the other hand to the narrowness of the pharyngeal cavity in the child, leading to more easy invasion of the orifice of the Eustachian tube by the inflammatory process. In consequence of this invasion the symptoms of aural trouble become the more prominent and direct attention rather to this part.

In the adult, acute naso-pharyngeal catarrh presents the ordinary symptoms of acute pharyngitis and coryza, which we need not recount here, in addition to which we find malaise, posterior cephalalgia, a heavy feeling in the head, tinnitus aurium, and slight deafness—all symptoms which are sometimes only the prelude to an acute otitis after having been the indication of an inflammation of the Eustachian tubes. There is usually little or no fever. The aural symptoms are unilateral or bilateral; they are sometimes entirely absent, which would seem to show that the tube does not in this case participate in the inflammation of the rest of the mucous membrane. We must

not, however, conclude from the absence of these symptoms the absence of extension to the tubes, for in adults we sometimes see a rupture of the tympanum with a purulent discharge without there having been any premonitory symptoms other than those of a slight acute rhino-pharyngitis.

There is a rather profuse secretion in the naso-pharynx of mucus, which is constantly swallowed or rejected, and by the dropping of which into the air passages the tracheo-bronchial mucous membrane may become infected. This secretion is not produced until the end of one or two days after the onset of the inflammation.

In the child the affection presents itself with somewhat more terrifying features. There is an attack of fever accompanied by chills, anorexia, thirst, sometimes vomiting; and slight convulsions may mark the onset of the disease. We also find the symptoms common to coryza and acute pharyngitis; but the most prominent symptoms, apart from the coryza, are the respiratory troubles (obstinate stridulous cough), and especially the symptoms of aural complications. Ringing in the ears, pain, and more or less marked deafness in one or both ears* usually open the scene, and often a few days later a discharge appears.

Nasal respiration may be abolished, and if the infant is nursing the taking of the breast may be impossible; it snores while sleeping, and often awakes suddenly in a fit of spasmodic, stridulous coughing, of a very terrifying nature, which continues until the air passages are cleared of the obstructing mucus.

In the adult, as in the child, the presence of an acute coryza with difficult swallowing and aural troubles should lead to an examination of the naso-pharynx. This examination, which is possible in the adult with or without cocaine, is very difficult of execution in the child. In the latter digital exploration is of great importance, but it ought to be avoided as far as possible until after the acute symptoms have subsided.

By means of posterior rhinoscopy we may see that the pus flowing down the pharyngeal wall comes from the vault of the naso-pharynx. Sometimes the palatine tonsils, the velum palati, its pillars and its posterior surface, participate in an inflammatory process and are of an abnormal redness; but this redness is always more pronounced in the naso-pharynx and beneath the mucous membrane lining its vault. The mucous membrane is no longer smooth but raised up here and there in little elevations, due to an isolated swelling of a closed follicle raising itself above the mucous membrane which itself is swollen.

* The deafness is not always recognized, especially if it be unilateral, the child hearing well with one ear and thus concealing the defect in the other.

Under certain circumstances one might see in the neighborhood of the pharyngeal bursa slight ulcerations involving only the epithelium (Mégévand).

The lips of the Eustachian tube are swollen and the orifice which they bound is sometimes invisible. The fossettes are covered with mucus like the rest of the naso-pharynx.

This examination, which cannot and should not be made in the child during the acute period, often requires in the adult the employment of the palatine elevator and cocaine anæsthesia.

Examination of the ears is very important. It shows both the condition of the tubes and the degree of participation of the tympanum in the naso-pharyngeal catarrh; for we find here all the signs of an acute inflammation of the middle ear with or without exudation.

Finally examination of the nose shows the presence of acute coryza.

Course, Duration, Termination.

Febrile symptoms of the onset, when they exist, are of short duration, even the period of full development seldom lasts longer than two three or days, and the disease tends to disappear very rapidly. Nasal catarrh sometimes continues longer than the other symptoms, so that when acute attacks follow one another frequently the nasal catarrh may presist as it were constantly.

The acute form of adenoiditis terminates either by resolution or by the passage into a chronic condition.

Complications.

Local.—The affection causes in the course of time, through its repetition, hypertrophy of the adenoid tissue of the naso-pharynx, analogous to hypertrophy of the palatine tonsils and to pharyngeal vegetations, which may close up the posterior nares.

Distant.—Leaving aside the troubles consecutive to chronic catarrh, of which we shall speak later and of which an acute adenoiditis is often the primary cause, the nasal and especially the aural complications are worthy of special attention. Chronic coryza, with or without hypertrophy of the mucous membrane, and otitis with all its consequences are what we must have in mind when we find ourselves in the presence of a patient suffering with acute adenoiditis.

Prognosis.

In itself the affection is not grave, but we need only recall the consequences of an otorrhœa to persuade ourselves that acute naso-pharyngeal catarrh should be carefully diagnosed and promptly treated.

Diagnosis.

Acute adenoiditis may escape detection in the child or at least may call attention to itself only when an aural discharge has occurred. However, when we see a child affected with this aural trouble we ought always to think of the most frequent predisposing cause, that is to say, of the presence of adenoid tumors in the naso-pharynx, and we should direct our inquiries of the parents in this direction. To the information on this point which the parents may give we should always add the testimony furnished by digital exploration, which alone enables us to affirm or deny positively the existence of adenoiditis.

When the above-mentioned symptoms exist in the adult, and an examination of the buccal pharynx and of the palatine tonsils has given negative results, we ought always to think of an acute adenoiditis. Indeed, in the adult this affection can be confounded with but three distinct diseases, viz., acute pharyngitis (commonly called catarrhal sore throat), acute coryza, and simple catarrhal otitis.

Acute coryza is accompanied neither by painful deglutition nor by ringing in the ears, still less by deafness and otalgia. Posterior rhinoscopy will show a normal condition of the naso-pharynx.

In simple sore throat the palatine tonsils and the velum pendulum palati are red and swollen. The patient has neither coryza nor aural trouble. This affection may, however, exist together with acute naso-pharyngeal catarrh.

Purulent otitis may occur without preceding lesions of the pharynx and of the Eustachian tube, but the previous history, the absence of adenoid vegetations, and the normal coloration of the mucous membrane will facilitate the diagnosis of the cause and will prevent us from accusing wrongly the retronasal cavity.

Treatment.

Acute adenoiditis being a catarrhal affection, it will be useful to prescribe at the beginning a general treatment consisting in the administration of a small dose of sulphate of quinine and of a mild purgative.

Our attention must, however, be directed chiefly to the local treatment, which should have three objects in view: 1. To subdue the actual inflammation of the mucous membrane; 2. To prevent the return of this inflammation by the removal of its cause; 3. To treat the complications. We shall consider more fully the two last-named objects when we come to speak of the treatment of adenoid vegetations, of suppurative middle-ear disease, and of chronic coryza. We shall

occupy ourselves here only with the measures instituted to cause disappearance of the acute inflammation, and by that means also to prevent its complications.

These means consist in inhalation, two or three times a day for four or five minutes each, of vapor from certain aromatic plants to which a head of poppy has been added. Into a vessel containing these aromatic plants is poured a litre of boiling water, to which may be added a teaspoonful of the following solution:

Essence of wintergreen, ...	3 drops.
Eucalyptol, ...	2 gm.
Menthol, ...	5 gm.
Pure alcohol, ...	105 gm.

In adults a warm retronasal spray of a slightly antiseptic liquid may be of some service. Nasal irrigations should not be practised, especially in the case of children, because of the obliteration of the choanæ by the hypertrophied and inflamed adenoid tissue.

The aural pains should be combated by mastoid counter-irritation (flying blisters, tincture of iodine, punctate cauterizations) and by sedative instillations such as carbolized glycerin, with or without atropine, and morphine glycerin.

Retropharyngeal applications of a two-per-cent. chloride of zinc solution, made at the proper time, that is to say when the disease begins to subside, will hasten resolution of the inflammation—a resolution which should always be total and should have existed for a certain time before we may attack the adenoid vegetations which were the primary cause of the inflammation.

Chronic Naso-Pharyngeal Catarrh.

(Catarrh of the Pharyngeal Bursa; Chronic Adenoiditis.)

Definition.—Chronic naso-pharyngeal catarrh (chronic adenoiditis) is a chronic general or partial inflammation of the mucous membrane lining the naso-pharynx. We need not give a separate description of Tornwaldt's angina or catarrh of the pharyngeal bursa of Luschka, for it has been shown that this bursa is merely a depression of the mucous membrane between two lobes of the pharyngeal tonsil; and this depression is not unique, for there are others lying more or less parallel to it, though less deep. Inflammation sometimes causes adhesion of the two borders of this central depression, a small orifice opening into a crypt being left; this is the entrance of the so-called pharyngeal bursa. Catarrh of this depression should not then be distinguished from that of the rest of the retronasal mucous membrane.

Etiology.

All the factors which may enter into the production of acute inflammation of this region may also determine the appearance of a chronic catarrh. Repeated attacks of acute adenoiditis are most often simply the external sign of a chronic inflammation during a period of exacerbation. We need not dwell then upon an enumeration of the conditions which favor or provoke chronic naso-pharyngeal catarrh. Special mention ought, however, to be made of climatic influences and in particular of atmospheric humidity. A season of thick fogs almost always is accompanied by exacerbations of the disease or by its appearance in persons predisposed to catarrhal troubles. It is frequently observed in America, and it tends also to become more and more frequent in Europe, especially in France. It is sometimes simply an extension of a buccal pharyngitis or of a chronic simple or atrophic coryza; the first is of rare occurrence, the second is much more frequent. In other cases it is chronic from the start, being provoked by the inhalation of air charged with dust or irritating vapors (naso-pharyngeal catarrh of copper turners, masons, plasterers, cigarette smokers, etc.).

The scrofulous and those suffering with various diatheses (gouty, herpetic, syphilitic, malarial, alcoholic) are most frequently attacked. Naso-pharyngeal catarrh is most common in children, a fact which ought not to surprise us when we remember that the eruptive fevers, very frequent at this period of life, are an active cause of the affection. The adult is, however, very often a sufferer.

Symptoms.

There are two forms of chronic naso-pharyngeal catarrh: the moist form, characterized by a more or less abundant muco-purulent secretion covering the surface of the mucous membrane; and a dry form, in which the secretions take the form of crusts. The first is seen most often in children and adolescents, in persons suffering with hypertrophic or simple chronic coryza; the second is most common in adults, especially in those who are the subject of atrophic coryza.

The disease manifests itself by a certain uncomfortable feeling or by slight pricking sensations which the patient localizes behind the velum palati; by an incessant desire, more marked in the morning, of freeing the posterior nares of adherent mucus which obstructs them, and by repeated efforts to clear the throat; by cough and sometimes even vomiting. These are the chief symptoms which call the attention of observant people to their condition, but which others less

careful of their person may experience for years without complaining, unless an acute attack should become grafted upon this chronic condition.

If the secretion is at all abundant the patient constantly feels mucus running down the wall of the pharynx, which causes him to make efforts to remove it – to scrape out the throat, if the expression may be allowed. The mucous discharge accumulates in the naso-pharynx during the night and becomes more or less dry, from which we may understand that the efforts at expulsion and the coughing fits are most frequent in the early morning. All the mucus does not remain in the posterior nares during the night, any more than all is rejected during the day upon its arrival in the buccal pharynx. That which flows down along the median line soon reaches the entrance of the œsophagus and is swallowed by an automatic movement of deglutition; while that which flows down the sides, after having filled the fossettes and come in contact with the lips of the Eustachian tubes, reaches the aryepiglottidean folds, guided there by the direction given to it by the posterior pillars of the palate. It covers this fold on each side, penetrates into the vestibule of the larynx, and would descend into the trachea did there not exist during the waking moments a reflex which provokes expulsion. This is the cause of the frequent hacking which is so tiresome to the patient and unpleasant to those about him. During sleep part of the mucus is swallowed, another part enters the trachea and bronchi and gives occasion to fits of morning coughing, followed by the expulsion of thick sputum, sometimes streaked with blood, which is simply the muco-pus fallen from the naso-pharynx, united with the product of secretion of the glands of the air tubes.

Deglutition is sometimes unaffected, sometimes, on the other hand, painful. In the latter case an examination of the pharynx suffices to determine the cause of the phenomenon: the muco-pus in its passage has given rise to the formation of pharyngeal granulations, and the continual irritation of those occupying the lateral surfaces of the pharynx, resulting from the contraction of the posterior pillars, is the cause of this painful dysphagia.

The voice is often modified, sometimes even lost, in consequence of the presence of muco-pus in the larynx, or of a small crust which has fallen between the two vocal cords. It becomes normal again only after an expulsive effort has dislodged this foreign body from the larynx.

If the inflammation has attacked the posterior surface of the velum palati the voice becomes more or less nasal in tone. On the other hand if the orifices of the Eustachian tubes participate in the catarrhal

trouble we often see, though in less severe form, the aural symptoms which accompany acute inflammation.

Chronic dry catarrh presents nearly the same subjective symptoms, although it appears as though the discomfort in the pharynx were greater and the prickings more marked. In addition there is a very distressing feeling of dryness, causing repeated efforts of deglutition and marked painful dysphagia.

Patients affected with one or the other form of chronic catarrh often have, especially in the morning, a disagreeable odor in the breath, a stale odor which is not that of ozæna. Some patients complain of occipital or cervical headache, always rather intense; but these pains have nothing of a pathognomonic character, as we find exactly similar ones in all acute or chronic inflammations of the nose or of the accessory cavities. Finally a certain number complain of sternalgia at the line of union of the manubrium with the body of the sternum.

Examination.—These various symptoms call for local examination, which may be made, according to circumstances, with or without cocaine, and with or without the employment of the elevator of the palate. The picture presented varies according as we have to deal with one or the other form of chronic catarrh. In the humid form we see more or less abundant whitish mucus flowing over the posterior wall of the pharynx and along the sides behind the posterior pillars. Granulations in greater or less quantity often dot the surface of the mucous membrane; they form sometimes a median and sometimes a lateral group. We shall return to this when we come to speak of the complications of the disease.

By means of posterior rhinoscopy we may follow the course of the mucous exudation up along the nasal pharynx, stopping at some point, or extending into the vault; we may often see it coming out from the median recess or opening of the pharyngeal bursa. In other cases, starting from the posterior and upper edge of the vomer, it stretches out in the shape of a fan along the grooves on the pharyngeal tonsil, joining there the secretion flowing along the pharynx. By turning the mirror in the proper direction we may note the presence of this secretion in Rosenmüller's fossettes and even on the lips and in the opening of the Eustachian tubes.

After the mucous membrane has been cleansed by the douche, spray, or a pledget of cotton it is seen to be reddened in places, with slight ulcerations here and there, and occasionally there is in certain parts a varicose condition. It is not very uncommon to find on the surface of the hypertrophied pharyngeal tonsil little whitish elevations, resulting probably from the accumulation of epithelial débris

in an obliterated follicle such as we see sometimes on the palatine tonsils. Pressure on the edges of the pharyngeal bursa, when it exists, frequently gives exit to pus.

In dry catarrh the naso-pharyngeal mucous membrane presents a shining varnished surface, without granular elevations; brownish-black or yellowish crusts are seen on the surface; the pharyngeal tonsil is little or not at all in evidence, and posterior rhinoscopy enables us to see in the posterior part of the nasal fossæ the lesions of atrophic coryza, analogous in all respects to those of the naso-pharynx of which they were the starting-point.

Complications.

We must regard as true complications of chronic naso-pharnygeal catarrh the acute exacerbations of the affection which give it all the characters of an acute disease with the consequences which that entails.

The pharyngeal granulations caused by the passage of the mucous secretion should also be regarded as one of the untoward results of the disease; they keep the pharynx in a condition of hyperæmia and themselves contribute to the formation of mucus and to the propagation of the inflammation towards the larynx.

The chronic inflammation and the desquamations, however superficial they may be, of the naso-pharyngeal mucous membrane furnish without doubt constant opportunity for the admission of pathogenic agents. Many infectious diseases, eruptive and otherwise, such as erysipelas, have often no other place of entrance.

The evil effects of the catarrh are by no means confined to the pharynx. The stomach often suffers from the presence of the mucus which has been swallowed, and many cases of gastritis are simply the direct consequence of the deglutition of naso-pharyngeal pus.

If we turn now to the organs of respiration we shall see that they also pay their tribute to the catarrh, commencing with the nasal fossæ, which seldom escape. Simple chronic or hypertrophic coryza may be the cause or the consequence of moist naso-pharyngeal catarrh; atrophic coryza usually exists prior to dry catarrh of the naso-pharynx.

From the naso-pharynx the lesion extends quickly to the pharynx and then to the larynx, where we may very often see the vocal cords reddened, and blackish and rather thick mucus covering various points of the laryngeal membrane. This chronic laryngitis with its acute exacerbations, especially in those who use their vocal organs to a considerable extent, in addition to its ordinary symptoms, is also complicated with tracheitis, which is perceptible on laryngoscopic examination. Bronchitis itself may ensue, and the patient who seeks advice only for his laryngitis and bronchitis often owes all these

troubles to the condition of his nasal and naso-pharyngeal mucous membrane.

We need not refer again to the aural complications which have been treated of sufficiently under the head of acute catarrh.

Finally, among the rare complications have been noted attacks of sneezing, reflex asthma (Heryng, Hack, and others), and a reflex nervous cough. We need only mention these accidents, which disappear usually with the catarrh which gave them origin.

Diagnosis.

We have here three questions to determine: 1. Is there a naso-pharyngeal catarrh? 2. What is its seat? 3. Is it primary or secondary to some other affection?

Adenoid vegetations or naso-pharyngeal syphilis or tuberculosis may be mistaken for the affection under consideration. The diagnosis may be established by the aid of the mirror and of the finger in the case of the first mentioned, and the associate buccal pharyngeal tonsillar and cutaneous lesions will enable us to affirm or reject the diagnosis of simple or symptomatic chronic inflammation.

Anterior rhinoscopy will prevent confusion with chronic coryza. We must also remember the possibility of a catarrh of the sphenoidal sinus and so avoid the mistake of taking this affection for a simple catarrh of the naso-pharynx. A posterior rhinoscopic examination will enable us to determine whether we have to deal with a catarrh of all the mucous membrane of that region or only with the so-called Tornwaldt's angina. If the latter be present we shall see the orifice of the median recess blocked with pus while the rest of the mucous membrane is normal.

In order to determine whether the catarrh be primary or secondary it will be sufficient in case of dry catarrh to practise anterior rhinoscopy in order to determine that the nasal lesion which is present was the cause and not the consequence of the inflammation of the naso-pharynx. If humid catarrh is present the question is not so easy of solution, for the coryza may as well be the consequence as the cause of the naso-pharyngeal catarrh. If adenoid vegetations are present there need be no longer any doubt, for they must be considered as the origin of all the evil.

Prognosis, Course, Duration, Termination.

The affection develops slowly and if we had not to take into account the complications, especially troubles of hearing and of digestion and disturbances in phonation, particularly in those who have to use the voice professionally, the prognosis would not be very grave.

But when we come to consider the disastrous consequences which may ensue during the course of the disease and the rebellious nature of the affection, we shall not be tempted to defer treatment, which, even if it does not lead to a rapid cure, may at least prevent the production of these complications.

Treatment.

The treatment is entirely local; it consists in removing from the naso-pharynx the mucus which accumulates on its surface and in modifying the mucous membrane by means of various local applications so as to prevent the reproduction of these secretions. It is often useful to resort to certain surgical measures which, in removing the cause, render the cure much more prompt.

The first condition is fulfilled by nasal, especially retronasal irrigations with alkaline liquids (solutions of bicarbonate of soda, of borax, or of sea-salt) and by direct applications or the use of the spray. We would emphasize especially the advantages of retronasal injections, the good effects of which we have often had occasion to observe. When made by means of a metallic cannula curved in an S-shape and provided at its extremity with a hollow bulb with numerous perforations, they have few of the disadvantages which have been said to belong to other methods (entrance of the liquid in the Eustachian tubes, cephalalgia, etc.).

After the surface has been thoroughy cleansed much good will be derived from the use of astringent powders, or, better, of a spray, carried directly behind the palatine vault, of a solution of carbolic acid, borax, glycerin, and water. These two operations should be repeated once or twice a day according to the severity of the case. The application of a solution of iodine to the mucous membrane will also be of service. The following solution may be used:

Trichloracetic acid,	0.25
Metallic iodine,	0.15
Iodide of potassium,	0.20
Wine of opium,	1.00
Glycerin,	120.00

or slightly caustic applications may be made, such as solution of nitrate of silver, 1 : 5, of chloride of zinc, 1 : 30 or even 1 : 15. The following formula is a very useful one:

Hydrochlorate of cocaine,	0.25
Pure glycerin,	5.00
Chloride of zinc,	1.00
Distilled water,	25.00

Surgical treatment will be indicated whenever there exist any follicular elevations, the last traces of the rudiments of adenoid vegetations, or a pharyngeal bursa. It consists in scraping away these projections either by one of the various forms of naso-pharyngeal curettes, or by the simple Gottstein knife, as if we had to do with true adenoid vegetations. Surgical treatment gives the best results of any, as well as the most prompt in all cases where it is indicated.

The treatment by natural waters (sulphur, chlorine, soda, or arsenical waters, or a sojourn at the seashore) may be employed according to the indications present in each particular case and according to the temperament of the patient, and when it is indicated by the state of the general health.

It is needless to add that when complications exist they must be treated according to the indications of each particular case.

Syphilis of the Naso-Pharynx.

The naso-pharynx is one of the seats of predilection of syphilis. It is not often that the primary lesion develops there, as may be imagined from the difficulty of inoculation; but the secondary lesions are quite frequent and the tertiary even more so. One thing worthy of remark is that the latter appear, in a great number of cases, in relation with hereditary syphilis. In such cases the lesions are seen comparatively early, in general between fifteen and twenty-five years of age, but when they are the lesions of acquired syphilis they seldom appear until between the thirtieth and fortieth years. We will now study the various lesions which characterize syphilis of the naso-pharynx in its different periods.

Primary Lesion.

The chancre is most frequently the consequence of inoculation by the contaminated finger of the physician who is making an examination of this region, or by infected instruments which are passed into the posterior nares, cases of which have been cited by Blanchet, Lancereaux, Moure, and others. Eustachian sounds have most often been the vehicles of transportation of the syphilitic virus. The physician's responsibility in such a case is very great, and we cannot insist too strongly on the care with which the finger should be cleansed in every instance before introducing it into the pharynx, where it is so easy to scratch the mucous membrane, even the most courageous patient often making involuntary movements through which the membrane is eroded. All instruments should also be thoroughly aseptic.

Chancre of the naso-pharynx is not common. It has, moreover,

often passed unrecognized because the possibility of such an accident did not occur to the physician as an explanation of symptoms which, except in one point, were in no wise pathognomonic. The symptoms were those of acute catarrh: the same obstruction of the nares and nasal intonation, the same pain on swallowing, the same aural symptoms, the same posterior cephalalgia, accompanied by a sense of weight and oppression (*céphalée gravative*). It should be noted, however, that the nasal obstruction is usually more marked than in simple acute catarrh, that the nasal intonation is more noticeable and of longer duration, and that the pain on deglutition is less severe. These differences might, perhaps, not be apparent were it not that the affection drags itself along tediously, and were it not for the presence of an important symptom in addition to those above mentioned—a symptom which, taken in connection with the difficulty of breathing, would suffice to indicate the existence of the trouble of which we are treating; it is never absent either here or in other parts of the body where a primary sore may be present. This symptom is glandular swelling which, in the case of a chancre of the naso-pharynx, is found in the neck at the angle of the jaw along the sterno-cleido-mastoid muscle, in the nucha, on one side or on two, according as the lesion lies in the median line or to one side. The rapid increase in size of the glands, the indolence of the swellings, their isolation one from the other are the characters which allow us to differentiate this condition from a simple adenitis. The swelling is in some cases very pronounced, so that the glands stand out prominently above the general surface; in other cases the condition is so slightly marked that it must be searched for to be discovered; indeed, even when the glands are greatly enlarged they may not produce any swelling on the surface.

The appearance of a roseola some days after, or its existence at the time of the first examination, would remove all doubt as to the nature of the condition.

It is probable that the appearance of a naso-pharyngeal chancre will not differ in any respect from that of a similar lesion of the tonsil, which appears according to Fournier under four very distinct forms: tubercular, ulcerating, anginous, and pseudo-membranous. Nevertheless the records of these examinations are scanty, the inspection having often been made at too advanced a period. The evolution of the chancre proceeds here in much the same way as in other parts of the body, but as a rule the accidents which follow it come on with greater rapidity and intensity; the malignancy of the affection seems to be increased by reason of the cephalic seat of infection, and also we may add by the absence of treatment in the early period.

Secondary Lesions.

We find here that the changes are exactly similar to those found on the pharyngeal tonsil and on the pillars of the fauces where like lesions often coexist.

The mucous patch in the naso-pharynx is more rare than that of the isthmus of the fauces, but it nevertheless exists (Moure and Raulin, Seifert). It appears as a grayish patch with rounded borders projecting above the mucous membrane, which here forms a narrow zone of a deeper red than that of the normal surrounding membrane. The eroded surface which forms the patch is smooth, red, very painful to the touch or when the neighboring parts are rubbed, and sensitive even to the passage of cold air. Below it there is a little infiltration of the derma (inflammatory œdema), the seat of which may give rise to complications analogous to those occurring in simple acute catarrh of the naso-pharynx, especially tubal catarrh and subacute catarrhal otitis. The mucous patch may occur on all points of the naso-pharynx, and we may even see it develop in the Eustachian tube or in the middle ear, thus deserving the term secondary otitis which has been given to this condition. Of all the naso-pharyngeal surface that part which is occupied by Luschka's tonsil or the closed follicles is, without doubt, the part most favored, if we may so express it, by the presence of the mucous patches.

It must not be supposed that this mucous patch is the only manifestation of secondary syphilis in the naso-pharynx; and hypertrophy of adenoid tissue of the region may be, independently of any trace of ulceration, a sign of infection of this nature. This hypertrophy is only temporary; it disappears ordinarily as a result of specific treatment; but it may persist for months if the patient is not put under treatment. It may cause all the complications consecutive to adenoid vegetations.

The cervical glands are swollen in this stage but less than when chancre is present. A careful search should be made for them.

Tertiary Lesions.

Gummata with consecutive ulcerations are the most common lesions of this stage; more or less vicious cicatrices are the consequence. We shall study both lesions. Tertiary syphilis may also attack the bones of this region, as elsewhere, and thus determine either the formation of sequestra, followed or not by the elimination of the dead bone, or gummy softening of the bone leading to its total disappearance with disastrous consequences. Finally these

different lesions unfortunately do not remain confined to the naso-pharynx, unless their progress be arrested by treatment. The lesions are prone to extend and we ought not to be surprised at seeing them invade the pharynx and nasal fossæ. A word concerning each of these accidents.

The *gumma* may be discovered before or after its ulceration. It is seated usually on the posterior pillars of the palate or at the level of the closed follicles which raise up the mucous membrane of the posterior wall of the pharynx. If it develops rapidly it occasions phenomena of posterior nasal catarrh, to wit, early occlusion, posterior cephalalgia, helmet pains, earache, dysphagia, regurgitation of liquids through the nose, nasal intonation. Nevertheless these symptoms may be absent or so little marked, especially if the gumma develops slowly, that they may escape the attention of the patient. This is true especially of circumscribed gumma, occupying the substance of the velum, which is painless and gives rise to disquieting symptoms only when it ulcerates; there may be at most only a slight nasal intonation, and yet if we look into the throat at this time we shall see the velum palati immobile in part and pushed forward on one side only. Sometimes a projection from its surface marks the presence of the tumor in its substance, but this projection is hardly appreciable except by posterior rhinoscopy. Up to the period of softening the mucous membrane preserves its normal color, and the physician is often not called upon to examine the lesion until ulceration has already destroyed a part of the velum.

The gumma instead of being circumscribed may be diffuse and involve a more or less extensive portion of the pharyngeal wall or of the velum. In the latter case there is infiltration of the entire organ, absolute immobility of the pillars, a wine-red color, especially of the posterior surface; the infiltration extends into the naso-pharynx, and it is to this form that the rapid development with the complications above described belongs.

Finally, as in the case of syphilis of the tonsils, but more rarely, we may observe a catarrhal form accompanied by fever and more or less grave general symptoms which might readily be mistaken at the beginning for a simple acute adenoiditis, which it resembles not only in its symptoms but also in its complications. Such an error will soon be made apparent by the gangrenous or diphtheroid aspect which the inflamed surface takes on and by the ulceration which follows.

Whether we have to do with the circumscribed or diffuse gumma or with the catarrhal form, we shall soon see ulceration set in in most cases, unless early and active treatment has been instituted. Oc-

casionally, however, the gumma proceeds rapidly towards sclerosis without the intercurrence of ulceration. This ulceration consists simply in the elimination of the products of degeneration of the cells which constitute the gumma. In order that this elimination may take place there must be inflammation and ulceration of the mucous membrane. In a brief period we find extensive loss of tissue involving the velum, perforating it or eating away its edges, sometimes destroying it completely, encroaching upon the posterior wall of the naso-pharynx and of the buccal pharynx, extending deeply as well as widely, attacking the lips of the Eustachian tube and destroying them, attacking even the vertebræ and the pterygoid processes at the vault of the naso-pharynx. The ulcerative process may even be observed to attack the lateral walls and open into the large vessels of the neck; the medulla itself may be exposed.

The appearance of these gummata differs in no respect from that of ulcerating gummata of other regions. The borders are serrated, raised above the surrounding surface; the base of the ulcer is sanious and secretes or rather passively exudes purulent matter of disagreeable odor. Indolence is one of their characteristic features, and they never give rise to any adenopathy. The odor from the throat and consequently of the breath of patients, in whom there is a sequestrum of the vertebræ or pterygoid apophyses, is very marked. In such cases also we may detect by means of the probe the presence of exposed bone surrounded by flabby granulations which secrete a large quantity of pus.

In cases of *necrosis* involving the sphenoid bone, I have always observed the presence of very severe lancinating pains referred to the fundus of the eye, diplopia, and often profuse suppuration, taxing greatly the strength of these patients who are much more numerous than we have been wont to suppose.

The employment of specific medication, when a sequestrum is present, causes no noticeable improvement and gives results only after the sequestrum has been expelled spontaneously or removed by the surgeon. When there is no sequestrum this treatment will speedily determine the diagnosis, if any doubt have existed, by bringing about cicatrization of the ulcer or rather of its borders, for the destroyed tissue is not reproduced.

Cicatricial Deformities.—The ulceration may heal even in the absence of treatment, but then at the price of certain deformities to which it will be well to refer briefly. So much cicatricial tissue may be formed in the naso-pharynx as completely to obliterate this cavity; the air no longer passes through the nostrils and the finger introduced behind the velum, which is ordinarily immobile, encoun-

ters no naso-pharyngeal cavity; it feels itself arrested by a hard fibrous tissue, cicatricial and permanent.

In other cases it is only the nasal fossa of one or both sides which is more or less completely obstructed, a condition which it is easy to recognize by the various means of exploration at our command. We may remedy this by means of progressive dilatation, or by the formation of a new orifice by the galvano-cautery and the maintenance of this orifice by means of dilatation until cicatrization is complete. There is no doubt that in these cases the narrowing takes place from the periphery towards the centre; it is an annular stenosis of the edges of the choanæ, the tendency of which is to approximate the borders and narrow the orifice more and more.

The cicatricial process more often involves the velum palati. The latter may be adherent throughout its posterior surface to the anterior surface of the pharynx and may thus shut off completely all communication between the pharynx and the naso-pharynx. The patient has an atrocious nasal intonation, breathes through the mouth, and in the course of time acquires the look of a child with large adenoid vegetations or some other obstruction to nasal respiration—in other words, the adenoid facies. Sometimes one side only is obstructed; the posterior pillar is exposed and stretched out as far as the median line.

The uvula itself may be free or adherent, situated in the median line or drawn to one side. In certain cases it is the only portion that is free, and the patient may by forced inspiration cause air to pass through the nasal fossæ, but in too small quantity to be of any use in respiration.

There is sometimes a considerable depression at the point of osseous insertion of the velum, a depression which is the result of an adhesion drawing in the pharyngeal surface of the velum. This part is also one of the points of election of ulcerating gummata.

In addition to these cicatricial deformities there may be others of every shape and form drawing the velum or the uvula upwards, sidewise, or backwards. The stenosis in these cases is caused not by agglutination but by the drawing together of the lateral walls—annular contraction.

Progressive dilatation in the treatment of adhesions of the velum is usually but partially successful. The attempt has been made to separate the posterior pillars and to interpose between the raw surfaces a piece of gutta-percha or some other substance, while cicatrization is taking place, in order to prevent the reproduction of the adhesions. We know, indeed, that the latter have a great tendency to be reproduced, and in many cases the result of the operations has

been negative despite the utmost precautions taken to prevent this very accident.

Diagnosis.—The diagnosis is to be made during the period of ulceration or after complete cicatrization has taken place.

In the first instance it is founded on the previous history of the patient, the indolence of the disease, the absence of adenopathy, and the progressive invasion of the neighboring parts. Tubercular and cancerous ulcerations have very different characteristics; the first take place in a late stage of tuberculosis, they are very painful and are of an entirely different aspect from gummatous ulcerations; the second, which are very rare, have indurated granulating borders, which bleed easily, break down under the nail, follow usually a lesion involving the isthmus of the fauces, and give rise to more or less marked dysphagia, while at the same time the patient's general condition rapidly deteriorates.

We need say very little concerning gangrenous ulceration, the symptoms of which are much more acute; the pain is marked, the odor of the breath is repulsive, the general condition is very grave, and the progress of the affection is exceedingly rapid. Then also the isthmus of the fauces is involved in this process much more often than the naso-pharynx.

In the presence of a cicatricial formation such as we have described the diagnosis ought never to be long in doubt, except as to the presence of a congenital malformation, which is seen but rarely, for the only other condition which could be thought of is a healed lupus. The congenital malformations are, as we have said, of exceptional occurrence in the naso-pharynx, except at the level of the choanæ of which they may cause the obliteration. There is, furthermore, in such formations none of the white cicatricial tissue which causes the deformity in the case of disease and which points to a previous inflammation. As this condition has been already referred to in the section on malformations of the naso-pharynx, we need not dwell upon it here.

Lupus is of slow growth; it eats away the velum gradually, attacking its free border; it gains ground in one direction while losing in another, and alongside of the ulcerating points we see little whitish cicatrices, evidence of a genuine process of cure. The uvula disappears gradually, the velum becomes rigid and is red in places, but the ulcerations which appear upon its surface are all superficial and do not involve the substance of the tissues, so that we may say with truth that most of the deep ulcerations of the naso-pharyngeal wall are syphilitic in nature and should be treated as such.

Treatment.—The question of treatment calls for but brief con-

sideration. We have here a tertiary lesion and iodide of potassium is the drug indicated. It may advantageously be combined with biniodide of mercury according to the following formula, which has always given us excellent results:

Biniodide of mercury,	0.15
Iodide of potassium,	15.00 to 20.00
Anisette,	100.00
Distilled water,	200.00

Of this mixture a tablespoonful may be taken morning and evening.

The surgical treatment indicated in the case of cicatrices of the naso-pharynx has been referred to in the section describing these lesions.

Tuberculosis.

As is the case with tuberculosis of the mucous membranes in general, that of the naso-pharynx presents itself under two distinct forms, viz., tubercular ulceration properly speaking and lupus—in other words, a rapid and severe form and a slow and relatively benign form.

Tubercular Ulceration.

Etiology.—Tubercular ulcerations occur only in patients who are already tuberculous and only at an advanced period of their disease. It is a fact which we have often noticed in regard to the buccal and nasal cavities that when a patient presents tubercular ulceration of the mucous membrane which lines these cavities he will be found to be profoundly infected with Koch's bacilli and his death is not far off. The naso-pharyngeal ulcerations are quite rare in proportion to the considerable number of tuberculous patients. They accompany lesions of the same nature in the larynx and in the buccal cavity and do not differ in their external aspect from those seen in the latter region.

Symptoms.—The pharyngeal tonsil is the favorite seat of these ulcerations. They begin by little cup-shaped erosions lying close to each other, then become confluent, forming deep ulcers with irregular borders. The presence of portions of undestroyed mucous membrane in the midst of the ulceration gives to it the appearance of a piece of cloth eaten by moths. The neighboring parts are sown with yellowish granulations in process of softening which contribute to the increase in size of the main ulcer. The base of the ulcer has the same granular appearance. The borders are generally a little infiltrated, which gives the appearance of a deeper ulceration than actually exists.

A single symptom sometimes betrays the presence of these ulcer-

ations, namely, pain. Its seat is variable; movements of deglutition refer it to the pharynx if the ulceration occupies the neighborhood of the velum, and especially of the posterior pillar. Nevertheless this pain may appear spontaneously or upon efforts of swallowing under the appearance simply of otalgia; this earache has in some cases been attributed to the presence of ulcers upon the lips of the Eustachian tube, but in other cases we are compelled, from the fact that the ulcers are situated at a distance from the orifice of the tubes, to refer it to reflex processes such as we observe in laryngeal affections. Earache may be the only symptom, but the pallor of the velum palati, which is rarely absent, is important in a diagnostic sense.

Diagnosis.—The presence of tubercular ulcerations of the larynx, pharynx, buccal, and even nasal cavities, as well as the evident condition of the lungs or other organs, will enable us to refer to its true cause an ulceration of the naso-pharynx, of which the external appearances, difficult to determine or to interpret, would not suffice to establish its tubercular nature. Finally, if we would be absolutely certain of our diagnosis we may scrape the base of the ulcer or take a little piece from its edges in order to examine it bacterioscopically.

Course and Termination.—What we have said above as to the general state of the patient and of the unfavorable conditions under which his organism is contending against its enemy, will dispense us from dwelling any further upon the course, duration, and prognosis of lesions of this nature.

Treatment.—This, in general, is very unsatisfactory, and the ulcer, in spite of all that we can do, will continue to invade the adjacent parts. Sometimes partial cicatrization takes place, but aside from the fact that this occurs rarely it has, in any case, no influence upon the general condition of the patient.

The treatment consists in antiseptic cleansing done very gently because of the pain provoked by contact of any body whatever with the ulcerated parts. The surfaces may be cleansed by means of a spray of some mild antiseptic solutions (carbolic acid, 1:1,000, for example, to which should be added a small quantity of hydrochlorate of cocaine), and the ulcers may be lightly touched with weak solutions of chloride of zinc (1:50) or of carbolic acid in glycerin (1:5 or 1:3). Antiseptic powders, such as iodoform, are also recommended for local application.

Lupus.

Etiology.—Lupus of the naso-pharynx is sometimes, though rarely, primary, and sometimes secondary to a lupus of the face, of the nasal

fossæ, or especially of the buccal pharynx. The lesions which are found on the uvula, the anterior pillars, and the anterior face of the posterior pillars are met with also on the posterior surface of these same pillars and of the velum which form a portion of the walls of the naso-pharynx. The other constituent parts of the naso-pharynx also do not escape the tubercular lesion.

Symptoms.—As in the case in which the disease involves the isthmus of the fauces, here also we meet with it in various forms. At the commencement there is a livid purple color of the surface showing invasion of the tissues below; the mucous membrane is smooth, dry, or granular and in certain places we see little opaline points, true precursors of the ulceration; in other parts there are little elevations above the surface of the mucous membrane. The affection soon presents itself under one or other of the following forms:

The new growth takes on the appearance called port-wine swelling; it becomes irregular, more marked in places, mammillated, with little ulcerations on the surface, and which may cause adhesions between the velum and the posterior wall of the pharynx, if they occupy the posterior surface of the pillars. The pharyngeal wall itself is not exempt; it presents the same granular projections and the adhesions become more and more firm as the process advances. The two adherent walls form one fibrous, cicatricial, and permanent membrane. This is one of the recognized modes of cure of tubercular processes. However, the cicatrization which is the result of the process in this region is none the less vicious and irregular, for at the same time that it causes in certain points agglutination of the two surfaces, the lupus attacks and destroys here and there the free portion of the velum, eats it away, and renders it unrecognizable.

In other cases ulceration is the predominant feature; it is preceded by the formation of pustules which rapidly break down and form a picture very like that of the ravages of tertiary syphilis in the pharynx, from which it differs, however, in the following points: its progress is much more distinct, the base of the ulceration is yellowish, granular, and sluggish; cicatrization occurs in certain points while the ulceration continues to advance in others; the epiglottis rarely escapes, and the same is true of the buccal pharynx and of the tonsils—in a word, the lesion is much more diffuse than is that of syphilis.

Adhesions of the pillars are of much less frequent occurrence in this form than in the preceding. Posterior rhinoscopic examination shows sometimes a complete destruction of the lips of the Eustachian tubes; at other times the swelling of the lips or the obstruction of the orifices

causes marked deafness after having first excited catarrhal or suppurative otitis accompanied by obstinate earache.

Course.—The lesion may attain its full development without the knowledge of the patient, especially when we have to deal with secondary lupus. It is, moreover, compatible with a condition of apparently excellent health. Its development is preceded in very many cases, especially when the lupus is primary, by repeated attacks of sore throat (Bazin, Desnos, Homolle). There is sometimes difficulty in swallowing, sometimes a nasal intonation appears suddenly and is the first symptom indicating the presence of an already fully developed lupus. The same fact is sometimes observed in syphilis.

Diagnosis.—In forming a diagnosis we have to take into consideration the age of the patient, his antecedents, and the appearance of the lesion. Sometimes it would be necessary to institute specific treatment before deciding whether we have to do with lupus or tertiary syphilis, either hereditary or acquired.

Treatment.—This is general or local, the first consisting in the administration of generous tonics, combined with small doses of iodine. It is, in a word, that of scrofula, on which it is needless to dwell.

Local treatment frequently gives excellent results, arresting the progress of the disease and hastening the process of cicatrization. It consists in applications to the affected parts of a solution of lactic acid or of chloride of zinc (1:50, 1:30 or even 1:10), made once or twice a week. Carbolic acid in glycerin solution (1:5 or 1:3) is also an excellent topical remedy.

The cicatricial contractions in the naso-pharynx consecutive to lupus are amenable to the same surgical treatment as those dependent upon syphilis, to which we have already referred.

Tumors.

Tumors of the naso-pharynx are benign or malignant. The first develop slowly, cause no inconvenience except by their volume, and do not become generalized. The second, on the contrary, are liable to recur, increase rapidly, and may cause the death of the patient as a result of cachexia or a generalization of the morbid process.

Among benign tumors are to be mentioned adenoid vegetations, enchondromata, papillomata, fibro-mucous and mucous polypi, and finally fibroid polypi, the latter being transition forms between benign and malignant tumors; for if they are benign in their histological characters and in the absence of generalization, they nevertheless act in many ways like malignant neoplasms.

Malignant tumors are much more rare; we may say that none is found in this region except fibro-sarcoma. Epithelioma of the isthmus of the fauces sometimes, indeed, invades the velum palati, the posterior surface of which forms part of the naso-pharynx; but it suffices for us to note this fact without delaying to describe the affection, which does not belong, strictly speaking, to the region now occupying our attention.

Enchondromata.

These tumors, which are of rare occurrence, are seated ordinarily upon the basilar process of the occipital bone, but, as in the case of fibrous polypi, they may also arise from the posterior border of the vomer. They may be primary, or secondary to fibrous polypi. Like these last-named tumors they send forth prolongations into neighboring cavities, having a marked predilection for the cranial cavity. These enchondromata are accompanied by the same symptoms as fibrous polypi, and give rise to the same deformities of the face; it is therefore needless to dwell upon these now, the reader being referred to the section in which the latter are described.

True primary enchondroma does not recur after ablation (case of Max Müller); the enchondroma following a fibrous polypus imitates the latter in its evolution (case of La Dentu, cited by Mackenzie).

The *prognosis* is favorable, the course of the affection is rather slow, and the means in our power for removing the tumor do not differ in any respect from those which we shall describe in the section on the treatment of fibrous polypi.

Papillomata.

Tumors of this nature are likewise very rare, only two cases being reported in the literature, namely, those of Sendziak and of Jurasz. One of these papillomata had its seat on the posterior portion of the inferior turbinated body and was adherent to no part of the naso-pharyngeal wall. The point of implantation of the other was not discovered. In each case mucous polypi of the nasal fossæ were also present. Although these tumors take their origin from the mucous membrane of the nose, they should nevertheless be regarded as tumors of the naso-pharynx, for they occupy its cavity and are manifested by symptoms proper to tumors of this region.

In the course of their growth these papillomata cause an irritation of the mucous membrane, which is manifested by naso-pharyngeal catarrh, with all its consequences. By themselves they may obstruct one or both of the Eustachian tubes and give rise to catarrhal otitis

and resulting deafness. Nasal respiration becomes impossible, and in regard to phonation the same troubles are observed as occur when adenoid vegetations fill the naso-pharynx. The velum palati is more or less displaced according to the size of the tumor, and the latter may even project through the isthmus of the fauces.

The surface of the tumor is pale in color and rugous, presenting the appearance of a raspberry; if it is touched with the finger it bleeds slightly; it is of soft consistence, but it is difficult to discover its seat of implantation while the tumor remains in place; it is easy, nevertheless, to determine that the pharyngeal vault is not affected (Sendziak).

Macroscopically the papilloma is seen to be divided into lobes, which in their turn are split up into rounded lobules, more or less projecting, giving to the tumor a close resemblance to a mulberry or a raspberry. Microscopically the tumor is seen to belong to the type papilloma with a small number of vessels.

The papilloma develops rather rapidly, but is nevertheless a benign tumor, causing no trouble except by its volume and the complications which may result from it.

It could hardly be confounded with adenoid vegetations, and even were the error made it would be unimportant, since the same treatment—namely extirpation—is applicable to both sorts of lesions. Adenoid vegetations are much slower in development, are more frequent in children, and are accompanied by the characteristic facies. Anterior rhinoscopy might possibly remove any chance of error by revealing the site of implantation of the tumor in the nose.

As regards *treatment*, the only measure deserving of mention is ablation. After local antiseptic irrigations of the nose and posterior nares, the surgeon should endeavor to include the whole of the tumor in the noose of the snare; the difficulty of the operation is precisely in the application of this loop, since we do not know where the papilloma is inserted. It will rarely be possible to place this snare through the anterior nares, at least without the introduction of the finger behind the velum. The buccal route will ordinarily be found to be the most accessible, but it is difficult to formulate any definite rules in this matter. The surgeon will combine his measures, employing, as may be indicated, the palate elevator, the laryngeal mirror, the finger, local anæsthesia, etc.; he may remove the tumor piecemeal if he cannot remove it entire—in brief, he will allow himself to be guided by the circumstances in each case. If we may judge from the cases reported, there will be no occasion to fear post-operative hemorrhage.

Fibro-Mucous and Mucous Polypi.

Definition.—By this term is meant benign tumors occupying the naso-pharynx, arising from its walls or in the neighborhood, the anatomical structure of which partakes of both fibroma and myxoma. These tumors, which are readily recognized to-day, since the histological analysis made by Cornil and Coyné, resemble much more closely mucous polypi of the nasal fossæ than fibrous polypi of the naso-pharynx. They borrow from the latter only that part of their anatomical structure, which gives them their consistence, and this would appear most probably to be due to the constitution of the mucous membrane from which they arise. The fibrous tissue may, indeed, be so little abundant that there may be no appreciable difference between these tumors and polypi of the nose, otherwise called myxomata. There are, nevertheless, true mucous polypi of the naso-pharynx which present all the characteristics of fibro-mucous polypi of the same region, and we need not dwell longer upon the distinction to be made between them.

Etiology.—According to some writers a chronic inflammation may give rise to the formation of polpyi, and Zuckerkandl goes so far as to say that mucous polypi are of inflammatory origin. This view appears to us to be exaggerated, and it is better to say that we are ignorant of the true causes of these tumors in the naso-pharynx, just as we are when they occur in other regions.

Fibro-mucous polypi are found in patients between sixteen and forty years of age; sometimes earlier than this, between the sixth and twelfth years (Moure), sometimes later, in the sixtieth year (Panas). Without being very frequent they are, nevertheless, more common than true fibrous polypi (Richet, Legouest, Labbé, Trélat, Mathieu, Degail, Panas, Moure, and others). They take their origin usually from the neighborhood of the choanal orifices, but they may start from the mucous membrane of the naso-pharynx at any point of its surface, more particularly in front of the basilar process of the occipital bone, on the fibrous bundle, which is also often a point of origin of fibromata of this region.

Symptoms.—The functional troubles experienced by the patient are identical with those accompanying other benign tumors of this region, especially papillomata and enchondromata. It will suffice, therefore, to mention them without dwelling upon them at any length; they depend entirely upon the seat of the polypi, their number, and their size. There may be obstruction of one or both nares, which may be complete or incomplete and which will become more marked

as the tumor increases in size. One fact worthy of remark is the obstruction of the nostril, which varies according to the position of the patient's head; this is due to the possibility of displacement of the tumor, a phenomenon dependent upon the existence of a rather long pedicle. The patient has a nasal intonation, there may be interference with olfaction, and it is not rare to note an obstruction of the Eustachian tubes, with its characteristic symptoms (noises in the ear, a sensation of fulness, deafness, catarrhal otitis) resulting from direct pressure.

On posterior rhinoscopy we see usually a rounded, grayish, translucent tumor on the surface of which run little vessels in different directions, more numerous in situations where the tumor is exposed to friction. The appearance is altogether like that of a polypus of the nasal fossæ, but upon palpation with the finger it will be found that the tumor has a greater consistence, a consistence varying in proportion to the quantity of fibrous elements contained in the polypus. Anterior rhinoscopic examination shows quite frequently the coexistence of mucous polypi in the nasal fossæ; but this fact is not constant, for frequently the choanæ alone are closed by the neoplasm of the naso-pharynx. Anterior examination enables us sometimes to see the point of implantation of the tumor, and it always enables us to see whether the tumor has invaded the neighboring cavities. There is no spontaneous hemorrhage nor does digital examination cause any bleeding, but it often permits us to determine the mobility of the new growth, provided its size is not excessive. These tumors vary in volume from the size of a walnut to that of a large hen's egg.

Course.—Fibro-mucous and mucous polypi have a constant tendency to increase, although slowly. They never disappear spontaneously whatever may be the age of the patient. But they are free from complications other than those due to the difficulty of breathing, of deglutition, or of hearing.

Prognosis.—This is favorable by reason of the benign nature of the tumor and of the fact that it does not become generalized, because it does not affect the general health, and finally, because of the slight tendency that fibro-mucous polypi have to recur after having once been removed.

Diagnosis.—The characteristic course and appearance of fibro-mucous polypi will prevent their being confounded with any other tumor of the naso-pharynx. If we add to this the consistence of the tumor, its appearance in persons of both sexes and at an age when fibrous polypi are not found, we shall have signs of quasi-certitude concerning the nature of the polyp. The fibrous polypus is indeed the only one concerning which there may be any error. It

is not rare to find tumors of this kind dumbbell-shaped, embracing the posterior portion of the septum, covering this on each side as if it were a saddle, a fact which renders their extraction difficult.

Although ordinarily smooth and of even surface, fibro-mucous polypi may be single or multilobed, but generally they are formed of one large cavity (pseudo-cyst) containing a certain quantity of watery or colloid fluid, which escapes sometimes spontaneously and then reaccumulates, or it escapes at the time of operation, greatly facilitating the latter. The knowledge of this fact is therefore useful in the matter of treatment.

Treatment.—There is only one therapeutic measure to be employed, namely, ablation of the tumor by means of the snare through the natural passages. There is no need here of any great preparatory operation, such as is often necessary in order to get at a fibrous polyp. The physician should employ simple means, but this is only repeating what we have said concerning papilloma, which is in a measure a polyp coming down from the nasal fossæ—we must be guided by circumstances. There are no fixed rules to be laid down; in one case, by passing the loop through the nose, we may seize the tumor and cut through its pedicle; in another case the loop should be introduced through the mouth and, guided if necessary by the finger, seize the polyp; in another case, again, it will be sufficient, when the pedicle is exposed to view, to divide it by means of the galvano-cautery, thus permitting the tumor to fall into the mouth and so be rejected (Moure).

If hemorrhage be feared (a very rare complication), it may be avoided by using the galvano-cautery loop instead of the cold wire.

It goes without saying that, before undertaking any operation whatever, the pharynx, naso-pharynx, and nasal fossæ must be painted with a solution of cocaine, in order to produce complete anæsthesia of the parts to be operated upon, a procedure by means of which the operation will be greatly facilitated.

We have referred to the absence of hemorrhage, but this is only in case the mucous membrane is not torn and if the polypus is taken away by the snare and not by the forceps or other instruments formerly employed for this purpose.

Fibrous Polypi.

Definition.—By this term we mean tumors formed of fibrous tissue, attached most frequently to the soft parts covering the basilar apophysis of the occipital bone, the characteristic of which is to send out prolongations into the neighboring cavities (nasal fossæ, maxillary and frontal sinuses, orbit, pterygoid fossæ, etc.).

Pathological Anatomy.—We will study successively the macroscopic appearance of the fibrous polyp, its anatomical formation with the modifications which this may undergo, its seat, the cavities which it invades, and the manner in which this invasion takes place.

It is of variable size, globular in shape, and as soon as it has acquired a certain size, sends out prolongations which take the shape of the invaded cavities; the naso-pharyngeal fibroid is red in its outer parts, hard, resistant, and very slightly elastic; if it be cut it creaks under the scalpel and shows a section which is smooth, yellowish, and uniform in appearance. In some of its prolongations it is not very unusual to meet with pseudo-cystic formations.

Under the microscope we clearly distinguish two parts, viz., a peripheral, which forms the envelope, and a central part forming the body of the tumor. The envelope is simply the mucous membrane of the naso-pharynx, but which has become thin in certain parts, tickened in others, and ulcerated in still others. The epithelium which covers it is no longer cylindrical and ciliated, as in the normal condition of the naso-pharynx, but is transformed into a protective epithelium composed of pavement cells with stratified layers on the surface and cuboidal cells in the lower layers. The mass of the tumor is formed of parallel bundles of dense fibrous tissue, the direction of which is perpendicular to the seat of implantation and which is occasionally wavy at the surface, giving rise to the appearance of peripheral nodules. Between these bundles we see a rather considerable number of cells in different stages of development, some being in the embryonal stage, the youngest; others, older ones, being larger, and others still having one or two prolongations. The proliferation of these cells and their growth cause the increase in volume of the tumor; their presence in large number gives to these fibroid tumors a very distinct character, inclining a little to the type of fibro-sarcoma.

The vessels of the mucous membrane are numerous and well developed; those which traverse the fibrous mass are much smaller; they possess walls which are, however, imperfectly developed, a fact which explains the hemorrhages that frequently take place during life from naso-pharyngeal polypi.

Occasionally, but very rarely, the tumor, after unsuccessful attempts at removal, has been observed to take on the characters of a sarcoma (Weber). Fatty degeneration, calcareous incrustation (J. Cloquet), and cystic degeneration (Maisonneuve, Cruveilhier) have also been observed.

The point of attachment of the tumor, always rather broad, lies in the great majority of cases (constantly according to Nélaton and his

school) on the soft parts which cover the basilar process of the occipital bone. There is normally in this region a fibrous layer of some thickness (18 mm. in thickness by 27 mm. in length, according to Tillaux) which is alleged to be the point of departure for the neoplasm. Although inflammatory adhesions may take place between the surface of the tumor and the mucous membrane in contact with it at any point of its surface, and although these adhesions may be mistaken for the original point of implantation of the fibrous polyp, it has, nevertheless, been shown recently that the tumor is sometimes inserted in some other part of the naso-pharyngeal wall, especially around the posterior orifice of the nasal fossæ, the internal wing of the pterygoid apophysis, and even the anterior surface of the cervical vertebræ.

In the process of growth the tumor rapidly encroaches upon the nasal fossæ; it may reach the accessory sinuses through the natural orifices, but this mode of penetration is not the only one employed by the tumor; it sometimes follows a more direct route by perforating the bones which separate the neighboring cavities, and thus gains entrance not only to the sinuses but to the zygomatic, the temporal, the orbital, the cranial, and even the buccal cavities.

Etiology and Pathogenesis.—Neither traumatism nor heredity has any part in the development of fibrous polypi of the naso-pharynx. The only two conditions which have any influence are age and sex.

The affection is met with most frequently between the ages of fifteen and twenty-five, and it is almost never seen in women. If it does occur in the latter it is usually after the age of twenty. We have recently had the opportunity of seeing a case of this nature in a woman upwards of forty years old. Fibrous polypi are occasionally seen in children under fifteen years of age, but the younger the child the less liable is he to suffer from this affection.

It is difficult to determine with certainty the pathogenesis, although a very simple hypothesis has been offered in explanation of it. Between the ages of fifteen and twenty-five years there are in the fibrous tissues in general, and especially in those in the neighborhood of the base of the skull, an increase of organizing activity which, according to most authors, would be a favorable condition for the production of the tumor, the latter being then only a deviation, with exaggeration, of this process. It is also admitted that the first menstruation in women produces a natural revulsive action towards the sexual organs and the mammæ, turning in that direction the excess of activity of the organism at this age.

Symptoms.—As in the case of most benign tumors, naso-pharyngeal fibromata may exist for a long time unperceived. The symptoms

at the onset have nothing characteristic about them, and the tumor is sometimes discovered only by chance upon a posterior rhinoscopical examination, made in what is supposed to be a simple chronic coryza.

The initial symptoms are usually a little nasal obstruction, slight difficulty of breathing, epistaxis, a discharge of watery fluid, sometimes more or less marked cephalalgia. There is nothing pathognomonic in these symptoms, and there may be no suspicion of the true condition until the day when the more marked signs which we are about to describe declare themselves. If an absolute impossibility of breathing through one nostril comes on, associated with a sensation as of a foreign body behind the velum palati, the patient is worried and consults his physician. A few well-directed questions will convince the latter that the nasal obstruction is not the only symptom experienced by the patient; the senses of taste and smell are already dulled, deglutition is frequently difficult, and liquids are sometimes rejected through the still-open nostril; epistaxis is more abundant and more frequent; the ear on the affected side has not been spared (sometimes both are involved) and there is a more or less marked unilateral or bilateral deafness, an evidence of obstruction of the tube, accompanied or not by otitis. But more than all, the patient carries the evidence of the disease in his voice; he has a strong nasal intonation and the correct pronunciation of certain consonants (*b*, *d*, *g*, *k*, etc.) is impossible. In certain cases he feels an almost irresistible desire to sleep (Whately) and this narcolepsy, which is more frequent at a later period, due probably to cerebral compression, is accompanied by a feeling of lassitude and sometimes by more or less intense neuralgia.

If the patient be examined at this stage we shall see the velum palati pushed strongly forwards and immobilized; the tumor in certain cases is visible behind the uvula, but this is not common. By means of anterior rhinoscopy, as well as by posterior, we may note the existence of a tumor filling the naso-pharynx, the surface of which is more or less red, sometimes covered by a sanious and fetid liquid, which also trickles down over the posterior wall of the pharynx. Upon retronasal palpation the finger impinges upon a hard body, very slightly elastic, more or less movable according to the size of its pedicle and according as it is fixed or not in the nasal fossæ.

It is sometimes possible to determine the seat of attachment of the tumor, either by the mirror or by the finger, the latter aided by a stylet passed through one of the nostrils; but as a general rule we cannot determine this with any certainty unless we can follow the tumor around in its entire circumference—that is to say, when it is not sufficiently voluminous to occupy the entire naso-pharyngeal cavity.

We should remember also that exploratory manœuvres must be carried out with great care, as the slightest touch determines in many patients profuse and even dangerous epistaxis.

If the tumor continues to grow we soon see new phenomena, pointing to its penetration into neighboring cavities. If, on the one hand, it extend into the pharynx, we see it encroaching upon the isthmus of the fauces, which it narrows, and it may interfere so much with deglutition at first, and later with respiration, that the patient is exposed to the dangers of asphyxiation. When it enters the nasal fossa from behind it quickly fills the cavity, dilates it, enlarges the space which contains it, wears away its walls, and finds exit where it can. It is in this way that the bones of the nose become separated, giving the nose greatly increased width, the orbital cavities become enlarged, the palatine vault depressed and its arch effaced. A little later we see more or less deformity of the face, the temporal and zygomatic fossæ are swollen out, the parotid hollow gives place to a more or less considerable swelling; the corresponding eye becomes more and more prominent, the patient suffers from diplopia and from epiphora due to compression of the lachrymal passages; the lids can no longer protect the globe of the eye, which is in a state of exophthalmos, and the appearance presented is that known as frog face (Mackenzie). The cornea becomes inflamed in consequence of irritation from the dust which is deposited upon it and which cannot be removed. The entire eye may be luxated or destroyed by a tumor penetrating the organ.

It is not uncommon to see the palatine vault perforated, the tumor breaking into the buccal cavity. The polypus sometimes projects from the anterior nasal orifices, showing there the same appearances as elsewhere, namely, a nodular and irregular surface, more or less red, ulcerated in some cases, and bleeding readily. In brief, all one side of the face is greatly increased in volume, prominent, and seems to advance so as to hide from the eye of the spectator the opposite side.

We referred above to penetration of the tumor into the cranial cavity. Gradual compression of the brain is betrayed by no symptoms in most cases other than somnolence and various sensory disturbances. When, therefore, we find such symptoms present in a patient suffering with a fibrous polyp, we should think of the possibility of a perforation of the base of the skull, an accident which is seldom recognized except at the moment of operation or on the post-mortem table.

Course, Duration, and Termination.—We have just passed in review successively the periods of onset, of growth, and of full development.

We have now to consider when and how the termination takes place. The younger the subject the more rapid is the progress of the tumor. It may take but six months to complete the first three stages, but ordinarily these require from eighteen months to two years, sometimes more.

The mode of termination varies in different cases. Unfortunately death is the most common ending; it is brought about by the repeated hemorrhages, by the increasing difficulty of respiration or of deglutition, by chronic poisoning due to the purulent liquid secreted by the ulcerated tumor, or finally, with signs of cerebral compression.

All cases are, fortunately, not so grave as this, and a cure may occur spontaneously in one of two different ways: either by gangrenous elimination of the tumor (Birkett, cited by Mackenzie, Duplay) or by its regression.

It is a fact that has been generally known for the last thirty years or so, that after the age of twenty-four or twenty-five years fibrous polypi of the nasal pharynx act like certain uterine fibroids after the menopause: not only do they cease to grow, but they even become smaller, perhaps to such an extent as absolutely to disappear. On the other hand, before this age it is very common to see them reproduced, after removal, with a desperate obstinacy, worrying both patient and surgeon, finally leading to a fatal issue. The transformation of a fibroma into a malignant growth would have the same result.

Prognosis.—The statement made above, concerning one mode of termination of the tumor, modifies in a certain measure the gravity of the prognosis, but only in cases in which the sufferer from a fibrous polyp of the naso-pharynx is in the neighborhood of from twenty-three to twenty-five years of age. The younger he is the more have we to fear for his life, for he will be more exposed to hemorrhages, to asphyxia, and to septicæmia, which rarely spare him. To these conditions, however, we may oppose the chances of surgical intervention which sometimes leads to surprising results. Under these circumstances the diagnosis of the affection cannot be made too early.

Diagnosis.—In its early stages a fibrous polyp of the naso-pharynx is often mistaken for simple chronic nasal catarrh. If the patient seeks advice at a time when he is suffering from partial obstruction of the nostrils or from aural symptoms the physician is often led to think of adenoid vegetations or mucous polypi. Digital exploration, however, readily enables him to detect adenoid vegetations, and when they are in a state to cause such inconveniences—an infrequent con-

dition in the adult—their presence will already have been suggested by the facies. All doubt may be removed by posterior rhinoscopy in such cases.

Mucous polypi are rarely single; they have a whitish, translucent appearance, like that which characterizes polypi of the nasal fossæ; they are implanted in the neighborhood of the choanæ, and their consistence is much softer than that of fibrous polypi. The fibrous polyp, usually single, slightly nodular, is covered by a red mucous membrane, seamed with large venules.

It is almost impossible to distinguish *in situ* a fibrous polyp from a sarcoma of the naso-pharynx; there are the same appearance, the same rapid development, the same hemorrhages; in such cases the age and sex are to be considered; the consistence of a sarcoma is less and superficial ulcerations are much more frequent.

Enchondromata are rare; their hardness makes them easily recognizable.

A case of neuroma of the naso-pharynx has been reported by Vacca Berlinghieri, but such tumors are so exceptional that they need not be taken into account.

Finally, the possibility of hernia cerebri need only be mentioned.

To know whether or not a fibrous polypus is present is well, but it is still better to determine its seat of implantation and the direction of its prolongations; it is important, especially, to know whether or not cranial penetration exists. In many cases it is impossible to determine the latter, for somnolence and coma are often absent, and atrophy of the optic disk, which has been regarded as a characteristic sign of cerebral compression at the base, is also absent in many cases; there is, therefore, nothing absolutely certain on which to base a diagnosis. We may add, however, that cranial perforation is happily exceptional, and we need not take its possibility into account unless in the presence of symptoms clearly pointing thereto.

Treatment.—This may be either palliative or curative. Palliative treatment consists in obviating the unpleasant symptoms of a fibrous polyp without attempting a radical cure. It is based on the fact of the decrease of the tumor and the possibility of its appearance at about the age of twenty-five years. If, then, the tumor does not arise until about the age of twenty or twenty-two years, we may, in certain cases, resort merely to palliative treatment. At this age it has been remarked that the fibrous polyp grows slowly, so slowly indeed that we may sometimes abstain from all treatment.

The palliative treatment is addressed on the one side to the nasal hemorrhages, and on the other to the interference with deglutition and respiration. The hemorrhages may be combated by plugging the

nasal fossæ and the naso-pharynx. We must avoid causing hemorrhage by abstaining from all unnecessary and imprudent manœuvres. Deglutition and respiration will be facilitated in certain cases by the removal of a portion of the tumor.

At the present day, when rhinoscopy has made such progress, we ought not to defer intervention in the young until the tumor has acquired so great a size as to cause considerable distress. It is much better to attack the evil at its incipiency. In that way we shall avoid many disappointments. Palliative treatment, therefore, should be reserved for those cases in which the tumor is already of large size or in individuals between the ages of twenty and twenty-five years. In all other cases radical treatment is preferable.

The curative measures are of two kinds; in the first the polyp is attacked through the natural passages, in the second the surgeon begins by creating an artificial passage which renders the operation upon the polyp itself much more easy of execution.

There are several methods of causing the disappearance of the polyp without forming an artificial opening. Cauterization by Canquoin paste, the seton, excision (Dieffenbach, Whately), exsiccation, crushing (Velpeau), compression (Bell, Malivern), and removal by the scissors (Borelli, Guérin, Bonnes, Hergott) need be mentioned only as a matter of record, not merely because of the difficulty of their employment, but also because of the accidents which they have caused and which have led to their disuse.

Avulsion is sometimes useful; it is done by means of strong forceps or by the fingers; but it is attended with the danger of fracture of the ethmoid bone or of subsequent cerebral meningitis or intracranial abscess. It has, however, the advantage that it often permits of removal of the tumor with its offsets without preliminary operation.

By means of the ligature we may, indeed, avoid hemorrhage, but its application is exceedingly difficult, the difficulty residing in the passage of the thread around the pedicle. If the tumor were of recent growth, if it did not fill completely or at least did not pass beyond the limits of the naso-pharyngeal cavity, a loop, preferably the galvano-cautery loop, might be passed around the pedicle by a skilful hand. This is a method which I have had occasion to employ twice with success in the early stages. Later the difficulties, the impossibility even, of applying the loop render this method impracticable. The écraseur of Chassaignac is less practical and exposes to greater danger of hemorrhage. The gradual ligature (Guillaume de Salicet, Rigaut, Hatin, Leroy d'Etiolles) exposes to great danger—danger of asphyxia at the moment of separation of the tumor, and

the danger of septicæmia and of hemorrhage when the eschar becomes detached.

Direct cauterization by the thermo- or galvano-cautery, although it does not give specially favorable results as regards the cure of the tumor, may at least render great service by destroying the pedicle and preventing the return of the growth once the tumor has been removed.

A method which is often deserving of great praise and which is applicable either directly or after a preliminary operation, consists in electrolysis of the naso-pharyngeal fibroma. Two needles in communication with the continuous current of fifty, sixty, or eighty milliampères are introduced deeply into the substance of the tumor, at some distance one from the other. At the moment of application there is a slight hemorrhage from one pole, but this is arrested almost spontaneously, especially if we take care to reverse the current before withdrawing the needles. The *séance* lasts for ten to fifteen minutes and may be repeated every fifteen to twenty days or every two or three months.

In order to watch the destruction of the tumor and even to apply the electrodes it will often be necessary to resort to one of the preliminary operations of which we shall now speak. When we do not wish or are unable to reach the tumor through the natural passages we may resort to one of three different operations: palatine, nasal, or facial. We must not forget, however, that these preliminary operations themselves are not free from danger and that several cases of death attributable to them alone have been reported. This furnishes another argument in favor of early operation and consequently of early diagnosis.

The palatine method invented by Manne in 1747 and modified by Dieffenbach (1834) and Maisonneuve, by Böckel and Nélaton, consists in a median and antero-posterior incision of the soft palate (Manne), in a simple buttonhole opening made in the same line and in the soft part of the velum (Dieffenbach, Maisonneuve), in transverse incision of the velum (Böckel), finally in antero-posterior incision of the velum followed by partial resection of the palatine plate of the superior maxillary bones (Nélaton). By this method, if the polyp is voluminous, we reach only its median portion and not its pedicle; if the polyp is smaller the opening is sufficiently large and the method is excellent. It should be mentioned that these operations on the velum are often accompanied by considerable hemorrhage, and it is well to reflect before undertaking them in patients who are already weakened by spontaneous hemorrhages such as often occur with fibroma of the naso-pharynx.

The nasal method consists in incision of the soft parts along the

dorsum of the nose and separation of the nasal bones (Verneuil), in turning the nose back over one of the cheeks after having made a superficial incision along the naso-pharyngeal fold (Chassaignac), or finally in turning the nose downward, after having performed vertical and lateral osteotomy (Ollier). The neoplasm is then taken away with strong forceps or the serre-nœud and, if necessary, the basilar apophysis may be scraped. Some surgeons, after having thus opened a way to the tumor, effect the destruction of the latter slowly, leaving the wound open during the necessary period. Although by this means a better result may be obtained as far as regards the neoplasm, hideous facial deformities are often the consequence. Most surgeons prefer to close the wound by first intention, thus obtaining a perfect esthetic result, although exposing the patient to a relapse of the tumor.

The facial method enables us to reach the polyp after total, or better, partial, after definite, or better, temporary, resection of the superior maxilla. Permanent resection enables us to destroy the tumor slowly and to see that it does not recur; temporary resection—a more brilliant operation, suggested by Huguier in 1852 and performed for the first time on the living patient by Langenbeck in 1859—avoids facial deformity and the troubles of mastication and of speech connected with a permanent resection, but, like Ollier's operation, it demands the entire destruction of the tumor at one sitting. It is well to add, however, that nothing prevents the surgeon from keeping the point of implantation under observation by means of the different exploratory methods at his command, and to repress, by means of the galvano-cautery, any attempted return of the tumor. The facial method, finally, does not cause such a disfiguring deformity as one might suppose; the soft parts supported by a prothetic apparatus, if the resection have been permanent, will often correct any deformity.

In certain cases, depending upon the prolongations and the size of the tumor, the facial method alone is applicable. But if a choice is possible, which it often is not, we would not hesitate to pronounce in favor of the palatine method, which, without leaving any external traces, enables us not only to remove the tumor but also to keep an active and direct watch upon the pedicle without the employment of any special instrumentation. The employment of the palatine method will also find its application when we have recourse to electrolysis, a method of treatment which is a little too much neglected in our day. An uranostaphylorrhaphy will remove, after the ablation of the tumor, all traces of the operation. This, in our opinion, is the operation of choice in cases of this kind, at least if the tumor has no tendency to become sarcomatous, which sometimes happens.

Hypertrophy of the Pharyngeal Tonsil.

(Adenoid Vegetations.)

Definition.—This term is applied to hypertrophy of the closed follicles seated in the mucous membrane covering the posterior surface of the vault of the naso-pharynx, and especially of those follicles which compose the glandular mass called the pharyngeal tonsil.

Adenoid vegetations and hypertrophy of the pharyngeal tonsil are but one and the same affection, so that we shall employ indifferently one or the other of these two terms. Czermak in 1860 and Voltolini and Loewenberg in 1865 were the first to speak of these tumors. Wilhelm Meyer in 1868 gave an excellent description of the condition, insisted upon its frequency, and gave it the name of adenoid vegetations. Since then many have occupied themselves with this question, among whom may be mentioned Guye of Amsterdam, Solis-Cohen, Thorner of Cincinnati, Woakes, H. Chatellier, Raulin, and the present writer.

Etiology.

Hypertrophy of the pharyngeal tonsil is common in all climates. According as the affection has become better known, certain countries possessing a dry and moderate climate, which appeared at first to be a condition unfavorable to the development of these hypertrophies, have been classed in the same category in this respect with the moist and cold regions.

Age has an undeniable influence, the affection being most frequent between the ages of three and ten years; below three years adenoid vegetations are rather more rare, but it would be a great error to maintain that the adult never suffers from them; on the contrary, he frequently suffers from them. It is proper to add, however, that the tumors begin in infancy, and that on the other hand vegetations, even when quite voluminous, give rise to serious symptoms much less frequently in the adult than in children; this is doubtless due to the larger size of the naso-pharyngeal cavity in the adult, so that there is free space between the tumor and the opposite walls of this cavity (Moldenhauer, Moure, Raulin).

Sex exerts no influence in the development of this affection. We cannot say the same of heredity. Meyer and Loewenberg have noted, and this is not exceptional, hypertrophy of the pharyngeal tonsil in several children of the same family. It might be more rational, however, to attribute these coincidences to a more general cause, one that certainly exists very frequently and is in its turn

hereditary, namely, lymphatism. In lymphatic children who have a tendency to hypertrophy of the tonsils, of the glands of the neck, and of the nasal mucous membrane, adenoid vegetations, which are themselves nothing else than an exaggerated development of lymphoid tissue, are of extreme frequency.

It has not been shown, notwithstanding recent attempts in this direction, that scrofula and tuberculosis are conditions favorable to the production of these tumors, or that the latter are often of a tubercular nature. The coexistence of adenoid vegetations and cleft palate (Meyer, Oakley Coles, Mackenzie) does not seem to me to be an evidence of the causative action of cold air and of food, for these same causes act daily on the palatine tonsil, which is similar in every respect to the pharyngeal tonsil, and yet hypertrophy of the former is far from being the rule.

If the predisposing causes which we have just passed in review are more or less doubtful, it is not the same with the determining causes, the action of which seems to be demonstrated. They provoke an irritation of the mucous membrane which finds expression in an hypertrophy of the closed follicles therein contained. Vapors and dust are certainly active agents in causing this irritation; the various simple and symptomatic naso-pharyngeal catarrhs are, however, the most common etiological factors; measles and whooping-cough are also in many cases to be regarded as causes of adenoid vegetations.

Pathological Anatomy.

Sections of adenoid vegetations differ very little from those of hypertrophied tonsils. We will examine successively the epithelium, the tissue proper, and the modifications which this tissue may undergo.

There exists normally at the surface of these vegetations and in the follicles passing into the substance of the tumor a cylindrical epithelium with vibratory cilia, consisting of one or two rows of cells only. These cells, placed side by side, are often interrupted by a little mass of six or seven mucous cells, forming utricles, radiating from a common point, a sort of little excretory canal. The neighboring cylindrical cells are crowded back by these mucous cells and the latter make no more than a slight depression on the basal membrane which supports them as well as the cylindrical cells. This disposition is very clearly marked on the portions of unaltered epithelium and especially on the walls of the crypts.

At other times the epithelium has proliferated and two, three, four, or ten or more layers are found; but the superficial cells have

become of the pavement variety, while the deeper layer constitutes a very beautiful row of cylindrical cells, and between these two forms many intermediate shapes are found. In a word, the epithelium has become a protecting epithelium and has taken on all the characters of that observed on the surface of the tonsils.

The basal membrane is scarcely visible, and the subepithelial tissue is seldom apparent except when inflammation exists. In the child the tissue proper of the part seems contiguous to the epithelium, from which it is very clearly distinguished. In the adult, if the vegetation has become indurated or is in process of atrophy, we often meet lines of connective tissue separating the substance of the tumors from the epithelial surface and even dipping down into the tissue itself, carrying there a number of vessels of a rather considerable size.

The substance of the vegetation is composed of a large quantity of closed follicles excessively developed and separated one from the other by a tissue of the same character but less close than that which forms the follicles. In sections colored with picro-carmine and under a low power we see only the rounded nuclei forming spherical masses, which differ from the neighboring tissues by the closer arrangement of their cells. Under a higher power we may see the adenoid network which serves as a support to the lymphatic cells and which contains a few scattered blood-vessels here and there.

In a large number of sections we have found, without being able to explain them, a number of little rounded masses, whitish in color, situated usually at the level of the closed follicles, in which the cells are scarcely colored on the borders and are not visible at all in the center. This would appear to be a partial degeneration of the tissue.

The presence of mucous cells, especially in the follicles, explains the possible formation of cysts in the pharyngeal tonsil. These rounded, smooth, more or less voluminous tumors, situated on the vault of the naso-pharynx, and which were thought to be primary, are found on closer inspection to belong rather to simple pathological products of a tissue which is itself pathological. We have had occasion to make sections of a cyst of this kind and have found it surrounded on all sides by closed follicles and limited immediately by an epithelium composed of cylindrical cells with vibratory cilia and mucous cells. The contents were mucoid in character and contained no traces of cells. A slight inflammation may have caused adhesion of the two borders of a follicle in order to produce the formation of a closed cavity which has gradually become extended.

There are certain follicles on the walls of which are found a protective epithelium like that on surfaces exposed to friction. The

closure of such a follicle at its mouth produces under these conditions, in the interior of the follicle, not a colloid cyst, but a white, pearly collection, formed of epithelial débris of semi-solid consistence, analogous in all points to what is observed in lacunar tonsillitis.

The transformation of adenoid vegetations into malignant growths has very rarely been observed. It goes without saying that the same pathological alterations are found on the surface and in the substance of this tonsil (syphilitic and tubercular lesions, catarrhal ulcerations, etc.) as are found in the buccal tonsil.

Symptoms.

To a practised eye the mere sight of a patient is often sufficient to enable it to be said whether or not he is a sufferer with adenoid vegetations. But the peculiar facies does not, as a rule, attract the attention of the parents, who may bring the child on a totally different matter. Sometimes they may have remarked that their child slept with the mouth open and snored, that his respiration was rough and broken in the midst of slumber by attacks of asthmatic dyspnœa, of croupy cough, or by true laryngismus stridulus. At other times an acute or chronic otorrhœa, a more or less marked deafness or earache, awakens the suspicions of the parents. If it is an adult who comes to seek advice, it is usually on account of a discharge from the ear, of a naso-pharyngeal catarrh, or a chronic stoppage of the nose. The physician must supplement the information furnished by the patient or his friends by his own examination, and with the exercise of a little care he will be able to build up, one by one, all the symptoms which denote the presence of vegetations in the naso-pharynx.

The facies is not peculiar to the affection which we are now considering, although it is called the "adenoid" facies, for it is found in all cases of obstruction of the nasal fossæ of some duration, produced by thickening or luxation of the septum, by large polypi, large tonsils, obliteration of the choanæ, cicatricial narrowing caused by adhesion of the velum to the pharynx, etc. Of all these causes, however, adenoid vegetations are the most frequently active.

The little sufferers from adenoid vegetations have a dull look, sometimes almost an idiotic appearance, their eyes are devoid of expression, are almost immobile, and the half-open mouth contributes to the formation of this characteristic facies; there is but little prominence of the cheek bones, the superior maxilla is undeveloped, the inferior maxilla and the lower lip are projecting—in a word, a pale, elongated face, without expression is that characteristic of a child suffering with adenoid vegetations.

The intelligence is usually perfect, although the mind is less alert, and if the child does not reply immediately to questions asked of him, the reason is usually because of the dulness of hearing due to the accompanying ear trouble. We cannot admit the unfounded opinion which would regard adenoid vegetations as a sign of degeneration (Régis). We must recognize, however, that there do sometimes exist defective memory, inaptitude for work, nasal aprosexia (Guye), or nasal cretinism (Raulin).

Nasal respiration is interfered with and may be entirely obstructed if the vegetations are medianly situated. The child snores in sleep, and besides the accidents mentioned above, which attract the attention of the parents, often suffers from nocturnal terrors and from profuse sweats. He is subject to sore throat, to earache, and to discharges from the ear. He presents the symptoms of a chronic naso-pharyngeal catarrh. He has frequent attacks of bronchitis or of broncho-pneumonia. Auscultation, performed while the child breathes through the open mouth, gives no special information, but if the examination is made when the little patient has his mouth closed respiration is heard to be faint and veiled (Grancher). The modifications of the voice are very interesting; the patient has a nasal intonation, the sound of the voice is obscure, and there are faults of pronunciation very appreciable by the ear; certain consonants cannot be pronounced; *m* is changed to *b*; *b* to *p*, children say Beyer instead of Meyer. This change is common in all who have a tumor of some size in the naso-pharynx. Troubles in hearing, especially frequent when the vegetations occupy the lateral portions of the pharyngeal wall, will be studied under the head of complications.

If we cause the child to open his mouth, we notice important changes: the superior dental arch no longer forms a regular semicircle; it constitutes a pointed arch of which the incisors occupy the centre. It is not rare to see two teeth developing in the same antero-posterior plane while their neighbors overlap each other more or less. The palatine vault, while giving to the superior maxilla such a configuration, is itself hollowed out in depth, taking the form of a pointed arch. The velum palati is often pushed forwards and becomes immobile. It is common to see at the same time an hypertrophy of the tonsils, narrowing the isthmus of the fauces, and in certain cases filling up all the space separating the anterior pillar from the uvula. The surface of the red mucous membrane of the pharynx, sown with granulations, is covered with muco-pus coming from the naso-pharynx.

The examination of the naso-pharynx with the mirror presents sometimes certain difficulties in the case of young children, while at

other times this examination is facilitated by the fact that the velum is pushed forwards when the vegetations are voluminous. When this examination is possible it enables us to see a nodular, irregular tumor of a cauliflower shape, pink in color, and often covered with a grayish mucous membrane; this tumor may occupy one or several places, but most frequently it is found at the vault and on the posterior wall. The tumors hang sometimes like stalactites behind the choanæ; at other times they fill up Rosenmüller's fossæ, obstruct the orifices of the Eustachian tubes, or stand out in little nipple-like projections from the posterior wall; they may also occupy the posterior surface of the velum, but this is more rare.

When posterior rhinoscopy is impossible or when we desire to get a more exact notion about the vegetations, we pass the finger, an aseptic finger, into the naso-pharynx; we may thus gather information as to the consistence of the growth, its exact position, and its size—information which is very useful when we come to operative interference.

Anterior rhinoscopy preceded by an application of cocaine may, in certain cases and to a practised eye, give some points useful in the way of diagnosis.

Finally, an inspection of the thorax and of the spinal column of the child will give evidence of the respiratory difficulty from which he suffers; the chicken-breast and the various spinal curvatures which have been noted by Lambron and Robert as a consequence of hypertrophy of the tonsils may equally as well be dependent upon the presence of adenoid vegetations.

Complications.

We have already spoken of acute and chronic adenoiditis following upon the presence of vegetations in the naso-pharynx; and we may also remember that pharyngitis, simple or stridulous laryngitis, attacks of spasmodic cough, bronchial catarrh, especially in damp weather, may have the same origin. Of all the complications the most serious are certainly those which attack the organ of hearing. They are produced in one of two ways: either by obstruction of the Eustachian tubes, especially in the child, or by direct extension of the inflammation, a not uncommon event in the adult. This is not the place to describe catarrhal otitis and its results as regards hearing; it will suffice to say that most acute or chronic inflammations of the middle ear in children have their origin in the naso-pharynx; the first symptoms are those of a tubal affection—noises in the ears, intermittent deafness—and these are followed by more or less complete destruction of the organ of hearing.

Diagnosis.

As a rule this presents no difficulty, especially if we take the precaution to employ the different methods of exploration which are in common use. In this way we shall avoid confusion with hypertrophy of the tonsils which often exists in enormous extent, with cicatricial narrowing of the isthmus of the fauces, or a more or less complete obliteration of the nasal fossæ, because of the adenoid facies which may be due to any of these causes.

Mucous and fibro-mucous polypi with their smooth, transparent appearance, their surface streaked with red lines, their rounded form, and their attachment by a pedicle will be readily distinguished. Fibrous polypi, with their prolongations, the epistaxis which they occasion, their course, their form, their point of attachment, and, especially their consistence, cannot be confounded with vegetations whose characteristics are so different.

Course and Prognosis.

After having increased in size for a certain period, adenoid vegetations remain stationary or may even become gradually reduced in size and finally disappear. This rule is far from being general, for we find adenoid vegetations in adults, even in those over fifty years of age, still acting as a cause of respiratory difficulty, and especially of deafness. Their presence may also cause serious inconveniences. Besides the arrest of development, of which they are in certain cases the sole cause, they often give rise to otorrhœa, of which deafness is only one of the least important results. Treatment therefore, and that speedy, is demanded.

Treatment.

By the very fact that adenoid vegetations tend to disappear spontaneously, or at least that with the development of the naso-pharyngeal cavity the inconveniences to which they give rise are no longer felt, all these tumors are far from being necessarily the subjects of surgical treatment. It will sometimes suffice, especially if the vegetations are small and occupy the median line of the posterior wall, to make irrigation of the naso-pharynx with a weak antiseptic solution or salt water to prevent adenoid vegetations from increasing. A general treatment by means of cod-liver oil, glycerin, phosphate of lime, sea baths, and a mild climate is advisable in the case of lymphatic children; nevertheless, neither general treatment nor any other medical treatment will cause the retrocession in any appreci-

able degree of true hypertrophies of the pharyngeal tonsil. Intervention is indicated upon the appearance of the slightest complication, but only after the subsidence of acute symptoms. But it is preferable to forestall these complications and to endeavor to remove vegetations which by their size or their situation would expose the patient to inflammation or ear troubles as a result of the slightest chill or exposure to cold and damp weather.

The surgeon may employ a number of means to remove these vegetations, either chemical caustics or the thermo-cautery or simple removal. Chemical cauterization is seldom employed at the present day. It would be made, after cocainization, through the nares or the mouth, but it has justly fallen into disfavor.

We may say as much of the thermo-cautery, which requires several séances, is difficult of employment in children, and offers no advantages over other methods. It often demands the employment of general anæsthesia and produces a cure only after the lapse of some time as a result of cicatricial contraction. It is better to resort to ablation.

A large number of instruments have been invented for the operation of removal of the pharyngeal tonsil. Among these we may mention the curved cutting-forceps, the surgical nail of Mottais, or sharp curettes, such as those of Trautmann, Hartmann, or Gottstein. Trautmann's curette is useful in scraping out the pharyngeal bursa when that is not very deep; that of Hartmann, having cutting sides, exposes the patient to wounds of the lips of the Eustachian tubes; Gottstein's curette, more or less curved according to the cases and more or less modified by different practitioners, is one of the most useful instruments; it enables us to operate quickly and to reach all parts. In consequence of the median notch intended to receive the edge of the septum we may reach vegetations situated in the vault and near the choanæ (Hicquet's modification). For operations on very young children, when the vegetation is single, median, and prominent, we may use with advantage the claw adenotome of Delstanche, with or without the writer's modification. Vegetations seated in the posterior part of the pharynx may likewise be removed with tolerable ease by means of Schultz's grooved adenotome, recently invented.

The following is the procedure which I employ and which has always given me excellent results: As far as possible during the days preceding the operation the region is carefully disinfected by means of irrigations of boric-acid solution, repeated two or three times a day. The child thus becomes accustomed to these irrigations, which will be of very great utility after the operation. I also

take care, when having to do with a child of from five to twelve or thirteen years, to have him subjected to a sort of training in order to accustom him to the passage of the adenotome. For this purpose an adenotome is introduced behind the velum and then withdrawn. It is rare that a child, even one not particularly docile, does not allow this instrument to be passed without the least difficulty after three or four séances. In this way the necessity for general anæsthesia is avoided.

The little patient is held on the knees of an assistant who imprisons the child's legs between his own. The assistant takes the hands of the child in his left hand, which he holds against his chest, and with the other hand he presses on the child's forehead, inclining the head slightly backwards so as to bring the mouth in a convenient position for the operation. For children from six to fifteen years of age two assistants are necessary, one to hold the hands the other the head.

The sterilized adenotome is then rapidly passed behind the velum, an operation which sometimes necessitates only depression of the tongue, at other times, especially in very young or refractory children, the employment of a mouth gag. The cutting surface is carried as high as possible against the posterior border of the septum and is then brought down, scraping backwards and downwards. Then, without withdrawing the adenotome, two or three lateral cuts are made in the fossettes in the same way as the median scraping. At the moment when the operator withdraws his adenotome the assistant rapidly depresses the child's head over a basin placed on the knees of the operator, and the vegetations thus fall out of the mouth. A tepid irrigation made immediately afterwards through one or the other nostril promptly arrests the hemorrhage, which seems at first to be abundant. One nostril after the other should be blown with some force; when the child is too much disturbed or does not know how to blow his nose an insufflation made by the Politzer bag will produce the same result. In general the bleeding does not stop until all the cut portions are entirely detached. It is sometimes necessary to make an irrigation with cold water in order to arrest this hemorrhage, which, however, seldom lasts for more than two or three minutes. Hard vegetations and those in the adult usually bleed more than soft vegetations and those in the child, a fact which has been noted also in the case of the tonsils. In the adult the vegetations are often interstitial and covered with a layer of mucous membrane, which is torn by the adenotome and sometimes bleeds profusely. In this case a second scraping by the curette becomes necessary.

A cotton plug is to be put in the ears, and the child should

remain forty-eight hours in his room, the parents being cautioned to apply a little blister to the mastoid process if he should complain of any earache.

As a rule the operation is not followed by any untoward symptoms. Cartaz has collected a certain number of cases of hemorrhage following removal of adenoid vegetations, but the accident is exceptional. If hemorrhage should follow the operation immediately we might first try irrigation with cold water or astringent solutions; but in case these irrigations should not suffice it would be easy to cover the adenotome with a thin layer of absorbent cotton or iodoform gauze and then reintroduce it. By this means pressure might be made for a few minutes, which would be all-sufficient to arrest the hemorrhage. In order to avoid hemorrhage Dr. Rousseaux, of Brussels, has recommended the employment of the electric curette, cutting through the pharyngeal tonsil gradually with the instrument at a dull-red heat.

It is not very uncommon to meet with suppurative otitis coming on four or five days after the operation. This accident may be prevented by using only sterilized instruments, having the field of operation thoroughly aseptic, and continuing the disinfection of the raw surface by means of antiseptic irrigations practised three or four times a day. If it were found necessary to curette the lips of the Eustachian tube or if they were accidentally wounded, we might have phenomena of tubal obstruction, first inflammatory and then cicatricial, leading in the end to more or less marked deafness. The frequent presence of vegetations in the fossettes exposes one to this accident.

When the vegetations are median, of large size, and when the velum is pushed forwards, it loses little by little its habit of contraction, so that in the days following the operation the nasal intonation may be even more marked than it was before the removal of the vegetations. It is well to inform the parents of this possible occurrence, which will disappear spontaneously at the end of some days, or still more promptly if electricity be employed.

Some operators employ the cold snare, or, better, the galvanic snare, passed through the nasal fossæ or through the mouth. Although by that means we may avoid hemorrhage, nevertheless there are many cases in which it cannot be employed. Simple scraping away of the vegetations by the finger is not to be recommended, because it is inefficacious and also exposes the patient to greater danger of infection. It may, however, be of some service when after the curetting it is found that some small vegetations, which could not be reached by the instrument, still remain in the fossettes. Of course such a procedure should be preceded and followed by an antiseptic irrigation.

It is useful to remember, when we have to deal with refractory children, and often even for the mere sake of rendering the operation more easy and more complete, that we may anæsthetize children in the sitting posture by means of very pure bromide of ethyl. This anæsthetic when properly administered often renders great service in short operations on the buccal or naso-pharyngeal cavity.

Malignant Tumors.

In this region, as elsewhere, malignant tumors may be primary or secondary or supervene upon benign tumors. They are rare, but they would perhaps be found less so if we made an histological examination of every neoplasm removed from the naso-pharynx.

The most frequently occurring malignant tumor in this region is the spindle-celled sarcoma, usually, however, associated with myxomatous, fibrous, or even cartilaginous formations (Stoker, MacDonald quoted by Mackenzie). The more rapid the growth of the tumor the more abundant is the infiltration of cells; the presence of some giant cells in the midst of the sarcomatous cells shows better yet the rapidity of growth. The vessels are numerous and have no proper walls of their own.

The *symptoms* of a malignant tumor of the naso-pharynx are the same as those of other tumors in this region; we need only mention nasal obstruction, tubal symptoms, the pushing forwards of the velum palati, the respiratory troubles, epistaxis, changes in the voice, continual coryza, and naso-pharyngeal catarrh. The discharge, however, which accompanies this catarrh is more abundant and more sanious than that of other forms and it often has an extremely fetid odor. Headache is not rare, especially at night, and earache is almost the rule.

Anterior rhinoscopy, after contraction of the mucous membrane of the nostrils as a result of cocaine application, enables us to see through one or both nostrils a rounded, more or less nodular, vascular, and reddish tumor. The neoplasm does not long remain confined to the naso-pharyngeal cavity, but soon invades the buccal pharynx, where it is readily seen on inspection through the mouth.

The consistence of the tumor is variable and depends in great part upon the greater or less quantity of fibrous or mucous tissue which it contains; this consistence therefore is of no special diagnostic value. There is no coexisting adenopathy.

By means of posterior rhinoscopy we may sometimes, in the early stages of the growth, be able to see not only the form and size of the tumor but also its point of attachment. Later this means of

exploration is of less value and must be supplemented by digital exploration.

Diagnosis.—Although the appearance of a malignant tumor will not enable us to differentiate it from other tumors of this region, the progress of the affection will enable us to judge of its character. Sarcoma grows rapidly and has a tendency to become generalized and to recur after removal. It causes general weakness, which soon advances to a cachexia.

Sarcoma of the naso-pharynx has many points in common with fibrous polypus of the same region. It will be remembered, however, that fibrous polypus is seldom developed in young people of from ten to twenty-two years of age, while malignant tumors are observed in all ages and in both sexes. The origin of a fibrous polyp is almost always from the posterior and superior wall, its consistence is very firm, and although it may return after removal it never becomes generalized; finally, it has a spontaneous tendency to cure in persons over twenty-five years of age.

The *prognosis* of sarcoma of the naso-pharynx is very grave, and an operation is always indicated.

Treatment.—We need not dwell upon the means to be employed for removing these sarcomata which are often pedunculated, referring the reader to the remarks concerning the treatment of fibrous or rather fibro-mucous polypi. If the ablation of the tumor be thoroughly done, it is possible in certain cases to obtain a definite cure, especially if the precaution have been taken to cauterize the point of implantation.

Neuroses.

We recognize at the present day various reflex troubles accompanying certain lesions of the nasal fossæ, such as attacks of asthma, of spasmodic laryngitis, etc., following changes in the pituitary membrane. These affections have received much attention, but the nervous troubles occasioned by alterations in the naso-pharynx have been less carefully studied. They have been noted by Heryng and especially by Baumgarten (1892). They consist especially in spasms, attacks of laryngismus stridulus (Ragoneau), whooping-cough, stammering, nightmare, etc. Even gastro-intestinal troubles (eructation, vomiting) and anuria have been noted as possible complications of chronic naso-pharyngeal affections.

We cannot agree with Baumgarten in classing among reflex troubles ringing in the ears and tubal or pharyngeal inflammations, affections which are sufficiently explained by direct propagation of inflammation to these different regions.

It is necessary to add that in all cases of reflex neuroses we must take into consideration the nervous condition of the patients who are thereby predisposed to reflex troubles and all the misery which accompanies them.

Treatment should always be at first general, because of this predisposing condition, and then local if it is found necessary to interfere. The latter, of course, must be appropriate to the condition originating the reflex.

DISEASES OF THE PHARYNX.

Malformations.

We have studied in the previous section the vices of conformation of the velum palati and we need occupy ourselves here only with those which involve the buccal pharynx and that below. Acquired deformities from tuberculosis, syphilis, or peritonsillar abscess will not detain us and we shall consider briefly only the congenital anomalies. These are of three kinds: according as they affect the pillars of the fauces, the conformation of the buccal pharynx, or the vertebral column upon which the posterior wall of the pharynx rests.

In some cases a separation of one or the other pillar from the lateral wall of the pharynx has been observed. The pillar is completely surrounded by mucous membrane; it is detached from the other portions of the velum, and at its free median portion, if the anomaly involves the anterior pillar, one may see the tonsil projecting between it and the lateral wall of the pharynx; if the posterior pillar is affected, the tonsil at the moment of contraction of the velum is seen to be resting upon the posterior wall of the pharynx between this pillar and the lateral wall. It is not uncommon to find in this case, as in the former, a symmetrical lesion, and this ought to make us think at once of a congenital anomaly. It is to be noted also that the perforation thus caused has not the cicatricial appearance of one due to an ulceration; the patient also denies any antecedent affection of the pharynx, and furthermore the lesion creates no functional disturbance.

Another malformation of the pharynx, which we have found also in the naso-pharynx, consists in a projection of the bodies of the vertebræ, forming either a ridge in the median line or a sort of hard tumor of all the posterior wall, pushing up the mucous membrane and narrowing the lumen of this cavity. This anomaly, which is often an acquired one (as in cervical Pott's disease) may also be congenital, and thus there is danger of error in the diagnosis.

Dilatation of the Pharynx—Pharyngocele.

Aside from the vices of conformation we may meet, in certain subjects having a narrowing of the œsophagus, with a more or less marked dilatation of a part or the entire circumference of the lower part of the pharynx. If the expansion is limited to a single point it constitutes a pocket or diverticulum formed of the mucous and submucous coats which pass through the muscular tunic of the pharynx, extending, generally, downwards and backwards between the vertebral column and the œsophagus. In some cases the diverticulum projects from one or the other side of the neck, forming a true tumor in this region.

The ordinary causes of this affection are the habit of swallowing greedily imperfectly masticated pieces of food, weakness of the walls of the pharynx, or the presence of small foreign bodies (the pits of cherries or other fruits) lodged in the lateral portion of the pharynx. Morell Mackenzie says that he has observed three cases in persons who have lived several years in the tropics and who showed symptoms of relaxation of other mucous membranes. Most of these patients had the habit of very rapid eating without sufficiently masticating the ingested food.

The *symptoms* of dilatation of the pharynx are very variable, sometimes almost *nil*, at other times sufficiently marked to attract the attention of the patient. These pharyngeal pockets may exist a number of years without causing any other symptoms than the return of food from the gullet as by a sort of rumination, and Morell Mackenzie cites the case of a patient in whom the dilatation had existed for fifty-one years without having had any effect upon his general condition. The patient generally has a sensation as of a foreign body in the œsophagus after eating, and inspection will sometimes reveal the presence of a soft tumor in the side of the neck which can be reduced on pressure, the undigested particles of food being forced back into the pharynx. When the diverticulum is full it empties itself little by little, and the patient sometimes rejects by regurgitation without effort the food which has been retained in this pocket. It may happen, however, that a piece will fall into the larynx, provoking attacks of spasmodic cough. The foreign body may even pass more deeply into the air passages and thereby lead to grave consequences. By means of the sound we may determine exactly the seat, the direction, the form, and the size of the diverticulum.

Pathological Anatomy.—Dilatation of the pharynx being a rare affection, we may readily believe that the records of post-mortem

examinations are not numerous. Rokitansky (*Archives générales de Médecine*, IX., 1840, quoted by Morell Mackenzie) had an opportunity of making a post-mortem examination on the body of one whose symptoms dated back forty-six years. He found a thickening of the mucous membrane of the posterior portion of the buccal cavity and œdema of the upper portion of the larynx; at the level of the inferior constrictor of the pharynx there was a diverticulum about 5 cm. long of the mucous membrane passing between the muscular fibres. This pocket was enveloped by the cellular tunic of the œsophagus in such a way that the pharyngeal canal opened directly into its cavity instead of opening into the œsophagus; if the attempt were made to pass the finger or a sound into the œsophagus it was impossible to avoid entering the diverticulum. The walls of the pocket contained some bands of pale muscular fibres, the œsophageal opening was narrow and the subjacent portion was atrophied.

Treatment.—The most certain means of cure is inflammation of the walls of the pharyngocele, with consequent obliteration of its cavity, but we must also remember that inflammation may lead to perforation of the walls of the pocket with extravasation of the food particles into the retropharyngeal connective tissue and all the accidents which may follow such a complication.

If the diverticulum is lateral the patient may, by compressing it during a meal with his finger or a suitable instrument, prevent its becoming so filled and thus avoid the discomfort or danger which may result therefrom. Also as soon as the meal is over he may be able, by a sort of massage or rather of taxis, to empty the diverticulum. A well-starched collar, with a soft pad, as recommended by Mackenzie, constitutes a very simple and practical apparatus.

If the opening of the pocket be visible in the laryngeal mirror we may endeavor to cause inflammation in it by means of caustic applications, and especially of the galvano-cautery. In extreme cases in which the patient is in danger of dying from inanition or suffocation we would be justified in opening the pocket from the exterior, excising it, and then reuniting the walls of the pharynx.

Wounds.

Wounds of the pharynx are comparatively rare. One would suppose that they might be more frequently encountered in connection with injuries of the throat, in attempted suicide or assassination; but even in these cases the pharynx is often spared, the wounding instrument generally being arrested at the trachea in the anterior half of the neck. They are, however, occasionally observed in cases of

homicide when the assassin's hand has struck with exceeding violence. Children, however, are those who are most exposed to wounds of the pharynx. A contused wound of the pharynx may result from injury caused by knives, pens, pencils, spoons, or other objects which the child may have been holding in the mouth when he happened to fall. Baratoux has reported the case of a child who had a wound of the pharynx inflicted by the stem of a pipe which he had in his mouth at the time of falling. Gunshot wounds of the pharynx have also been noted.

Symptoms.—The main symptom at the beginning is hemorrhage, the extent of which will vary according to the size of the vessels and the part of the pharynx which may have been wounded. It will be readily understood that a wound of the lateral wall may cause a violent, even a fatal hemorrhage. At other times there may be a simple oozing of blood, moderate in amount, or there may be a submucous effusion of blood which, later, results in inflammation with the formation of an abscess.

The swelling in most cases is accompanied by severe pain, which is felt especially upon deglutition. In some cases liquids may be returned through the nostrils. Occasionally the movements of the neck are much restricted, especially when the wound is of some size.

The symptoms may subside quickly or there may arise a considerable local œdema suggesting an abscess. If the lesion involve the upper part of the pharynx we may have earache or even acute middle-ear disease with consecutive perforation of the tympanum, just as occurs in certain cases of inflammation of the posterior nasal fossæ.

Diagnosis.—The question of diagnosis need not detain us, for wounds of the pharynx will be readily recognized on simple inspection of the throat after cleansing the parts.

Prognosis.—This varies according to the extent of the wound, its depth, and also the part involved. If there is much serous effusion, we must consider the possibility of an acute inflammatory process and consequent production of an abscess. The hemorrhages may, by their abundance or their frequent recurrence, influence the prognosis unfavorably. Wounds resulting from external violence are, as a rule, less serious than those produced from within.

Treatment.—The first thing that calls for attention is the hemorrhage. If it be not too abundant and if it come from a point in the median line, simple digital compression or pressure by wads of lint or cotton will suffice to arrest the flow. If, on the contrary, there is a wound of one of the arteries in the lateral wall of the pharynx, it may be found to be absolutely necessary to ligate the carotid. A

case has been reported (Morell Mackenzie) in which the epiglottis had been nearly cut away. In such a case it would be necessary to resect the organ, which might otherwise become detached and fall into the larynx. It could be removed by either the bistoury, the epiglottome, or the galvanic loop.

When resulting inflammation of the pharynx has extended to the larynx we must resort at once to tracheotomy. When, following extensive lesions of the pharyngeal wall, deglutition is very painful, it will be necessary to nourish patients through the stomach tube. The inflammation must be combated by emollients and the wound cleansed with antiseptics. Finally it will be necessary to watch the case most carefully in order to prevent possible adhesions and cicatricial contractions of the pharynx.

Burns.

It is especially in England and America that burns of the pharynx are encountered. This is due to the deplorable habit of leaving within reach of children kettles containing boiling water or very hot tea. The children, in their efforts to drink from this vessel, often receive extensive burns of the mouth and pharynx by the boiling liquid. This being one of the most frequent causes of burns of the pharynx, the injury is consequently met with most frequently among children. Similar lesions are observed in persons who, through accident or with suicidal intent, swallow caustic substances, such as sulphuric acid, caustic potash, and the like.

Symptoms.—The first symptom is a severe pain in the pharynx radiating to the ears, which is felt upon swallowing, especially in cases in which the burn has seriously involved the epiglottis, the base of the tongue, or even the entire œsophagus.

Any respiratory symptoms that may supervene are due directly to the swelling of the regions affected. If the lower portion of the pharynx have been wounded the epiglottis will probably be swollen and consequently the symptoms will be of considerable severity. If, on the contrary, the upper part of the pharynx alone is injured the respiratory symptoms will be but slightly if at all marked.

The local symptoms vary according to the nature of the offending substance. If this is a boiling liquid the parts affected will be covered with a whitish pellicle, and at the same time there will be swelling due to serous infiltration of the mucous membrane. As a result of the action of corrosive poisons, such as sulphuric acid, for example, we find a brownish scar, and the outer layers of the mucous membrane are more or less detached according to the degree of

concentration of the caustic; at other times the membrane is detached like a ribbon rolled upon itself in the form of a cylinder. If any vessels are visible they will be seen as blackish lines, the blood being the color of shoe-blacking. The pains may assume a very considerable intensity.

When the burn has been caused by nitric acid the eschars are of a yellowish or lemon color; they are not quite so deep as those resulting from the action of sulphuric acid and they are rapidly cast off. The pain in a burn of this kind is of comparatively short duration.

Hydrochloric acid causes blackish scars. The surrounding mucous membrane is at first of a bright-red color due to inflammation; if the burn be deep the mucous membrane may be softened or even pulpy in consistence.

Oxalic acid causes little whitish scars and softens the mucous membrane, which becomes friable; the coagulated blood in the vessels gives them the appearance of black lines.

The scars resulting from the action of carbolic acid are also whitish; the mucous membrane has a shrivelled appearance. The pellicle which forms is rapidly thrown off.

Among the alkalies we may mention more particularly caustic potash and ammonia. The first of these substances forms brownish or blackish eschars like those resulting from the action of sulphuric acid. If it be taken in diluted solution the eschars are yellowish or of a pale yellow color. Ammonia acts in the same way, but it produces less deep effects and a less intense congestion of the parts; its eschars are blackish.

Pure phosphorus produces simple slate-colored patches of the buccal mucous membrance. The breath of patients who have taken phosphorus has a very characteristic garlicky odor (Peter, Morell Mackenzie).

Tartar emetic causes burns analogous to those resulting from its application to the skin. Disseminated pustules corresponding to the points of application of the caustic are seen on the mucous membrane of the pharynx. The patient complains of a very marked metallic taste in the mouth. Later blackish crusts are formed with exfoliation of the mucous membrane which appears gouged out in places.

Chloride of zinc in concentrated solution forms a very caustic liquid, which produces whitish or rather grayish scars of considerable depth with destruction of tissue.

Corrosive sublimate and other salts of mercury produce a sensation of dryness with pains at the back of the mouth. The mucous membrane is eroded and superficial ulcers result. There is also accompanying mercurial stomatitis.

Nitrate of silver leaves the whitish pellicle familiar to all; it also causes a metallic and astringent taste in the mouth.

Iodine produces a yellowish-brown discoloration which disappears rapidly, with or without leaving any traces, according to the degree of concentration of the liquid.

Salts of lead occasion a simple irritation of the pharynx (the same is true of iodide of potassium) which may pass on to a slight inflammation, sometimes œdematous, of the parts (uvula and aryepiglottic folds).

The course varies greatly according to the nature of the agent causing the burn—its caustic power and the depth and extent to which the tissues are involved. If the burn have been slight the progress will be rapid and recovery prompt. If, on the other hand, the burn have been deep, the casting-off of the eschars will take considerable time and the healing process will be correspondingly tedious. Finally, very extensive burns may cause serious troubles, such as œdema of the glottis or perforation of the œsophagus or stomach, with all the serious consequences of these accidents.

Diagnosis.—This will usually be made from the history of the case, especially if the ingestion of the caustic have been voluntary and with suicidal intent. The vomited matters should always be examined, and, if possible, we should get possession of the bottle which contained the liquid. The determination of the nature of the caustic is sometimes difficult, since many of the eschars resemble each other, and often the patient is not seen until some time after the accident, when the lesions will present nothing characteristic.

The *prognosis* will vary according to the extent of the burns and their depth. We have just said that there may be an œdematous infiltration of the pharynx and larynx, a complication which will affect unfavorably the prognosis, and a favorable opinion should not be given until the œdema has disappeared. If the œsophagus is involved the prognosis may be rather grave. Finally, we must bear in mind the possibility of a resulting stenosis of the pharynx or of the œsophagus which may affect unfavorably the prognosis in any given case.

Treatment.—If called to see the case early the physician should prescribe, or, better, should himself perform, very thorough lavage of the mouth, pharynx, and stomach. He should also administer the appropriate antidotes—alkalies, when acids have been ingested, but milk or pure water in case of poisoning by caustic alkalies. One danger of lavage is that it may lead to perforation of the softened walls of the œsophagus; but we may have the consolation of knowing that if this canal ruptures easily under the pressure of an œsophageal

sound carefully introduced, we have merely anticipated a perforation that would inevitably have occurred soon in the natural course of events.

If the lesions are slight we should prescribe emollients in the form of gargles or vapor, or applied very carefully with a brush or pledget of cotton. If the pain on swallowing is severe excellent results may be obtained by painting the parts with morphine or cocaine, or if necessary with a combination of these two local analgesics. The food, I need hardly say, should be administered through a stomach tube. Later we may replace the emollients by astringent applications.

If the burn results in the formation of a stricture of the pharynx through adhesion of the pillars to the posterior wall, we must make daily progressive dilatation. We should, however, not forget that complications of this kind are often difficult to avoid and we may sometimes be obliged to resort to surgical measures for their relief. Stricture of the œsophagus due to this cause is to be treated as other strictures of this canal.

Gangrene.

(Gangrenous Angina.)

Gangrenous pharyngitis may be primary and confined exclusively to the pharyngeal wall, but most frequently it attacks first or simultaneously the tonsils and velum palati. It may be consecutive to a general alteration of the blood or to a violent inflammation of the pharyngeal wall. This sort of pharyngitis has been studied by Trousseau, who regarded it as analogous to noma. Rillet and Barthez have also given an excellent description of it in their treatise on the diseases of children.

Symptoms.

Gangrenous pharyngitis is announced by an intense fever with violent sore throat. The initial inflammation, characterized by great redness, is soon succeeded by the formation of an eschar of variable size. The patients fall into a state of collapse and of prostration analogous to that of cholera. The temperature falls, the pulse becomes slow, falling sometimes as low as fifteen to the minute (Gübler). This state is accompanied by pallor with discoloration of the integument; numerous petechiæ are present, indicating the profound alteration of the blood. It is not rare to see at the same time a fetid diarrhœa, with or without hemorrhage, denoting the participation of the intestinal tube in the infectious process. Other symptoms observed by Trousseau were diplopia and superficial phlebitis.

The fetid odor of the breath is very characteristic. The expectoration may be mucous, muco-purulent, or wholly purulent in character; it has a fetid odor analogous to that observed in gangrene of the lungs with expectoration of mucous débris. The fetor is generally characteristic of the affection, a fact to which Ramón de la Sota, of Seville, has drawn special attention.

Examination at the beginning of the disease shows redness of the pharyngeal wall, of the tonsils, and of the pillars of the fauces. Soon we find eschars forming upon blackish points; these are followed by very irregular ulcerations with notched edges of a blackish or grayish color, from which exudes a fetid sanious discharge. At other times the lateral walls and the anterior and posterior pillars are affected, the entire mouth may also be more or less invaded, so that we may then regard the case as one of noma. The larynx and œsophagus are also invaded in certain cases, when we may observe more or less generalized desquamation of the mucous membrane and extensive ulcerations, which may cause death through blood changes, defective alimentation, or hemorrhage (Ramón de la Sota).

In all cases in which the affection has progressed to a certain stage repair is very slow and prolonged. If the muscles have been exposed the patient rapidly succumbs.

Gangrene of the pharynx often terminates in death. This fata issue may be due either to heart failure or to pulmonary or intestinal hemorrhages. In certain cases suffocation supervenes with frightful rapidity. If the eschars are thrown off we may see as complete a cure as is possible take place, but this mode of termination is perhaps the least common.

Prognosis.

We shall appreciate the gravity of the prognosis when we remember that Trousseau and Morell Mackenzie had only one or two per cent. of recoveries. The disease is a serious one, not only in itself, but in all the complications which it may cause on the part of the lungs, of the nose, of the intestine, of the œsophagus, and of the larynx; and the general condition of the patient, which is usually nothing to boast of, also adds to the gravity of the prognosis.

Diagnosis.

Besides the odor, which is almost sufficient in itself to characterize the disease, we believe that the other symptoms can hardly leave any doubt as to its nature. The only affection with which gangrene of the pharynx could be confounded is diphtheria; but in the latter

the patches are whitish, there is besides an enlargement of the glands of the neck, and the symptoms are less grave. The diphtheritic patches are situated rather on the lateral walls of the pharynx. Finally, in doubtful cases a bacterioscopic examination will suffice to remove any doubts which may exist.

Treatment.

In the presence of such a grave affection as gangrene of the pharynx, it is evident that we must act with considerable energy. We should, in the first place, endeavor to maintain the strength of the patient by means of generous tonics, wine, champagne, cognac, and cinchona. Indeed, quinine would seem to be especially indicated here.

Locally the affection is to be treated by means of antiseptics, and for this purpose we may employ carbolic acid, thymol, salicylic acid, or resorcin, and chloral may be of signal service. Washes of lemon juice, lactic acid, thymol, iodoform, or iodol may be of service or a useful application is carbolized glycerin, 1:10, 1:5, or even 1:3, provided that the physician make the application himself. Applications of a solution of chloride of zinc, 1:30 or 1:15, may also be of service. In certain cases we might try again hydrochloric acid, or the perchloride of iron, formerly recommended by Trousseau and Gübler. These different applications may be employed in the form of douches or sprays, or as direct applications on a brush or pledget of cotton. The thermo-cautery has also been advised, but it is perhaps not without its disadvantages.

We must avoid making scarifications or incisions in the diseased tissue, the borders of which might slough and cause death by hemorrhage, as in a case reported by Ramón de la Sota.

On the appearance of laryngeal complications we must resort to intubation of the larynx, which would appear to me to be specially indicated in these cases, the œdema being of a temporary nature, or to tracheotomy, incision being made in the crico-thyroid space or directly into the trachea, according to the indications in each case.

Peripharyngeal Phlegmon.

By this name is understood a circumscribed or diffuse phlegmon occupying the cellular tissue surrounding the pharynx.

If we remember that the pharynx has, in its upper two-thirds, the shape of a gutter, opened anteriorly, the convexity of which is in contact with the vertebral column, and that in its lower third it forms a complete canal, we shall be in position to accept the following state-

ment: in the buccal and nasal portions of the pharynx we have to do only with retro- and latero-pharyngeal phlegmons and abscesses; in the laryngeal portion we may meet sometimes with anteropharyngeal abscesses as well. An abscess which is primarily retropharyngeal may indeed invade the cellular tissue of the lateral portions of the pharynx, but this is not the rule, and the difference of symptoms characterizing phlegmons in these two localities calls for the distinction which we shall make in their description. We will study first

Retropharyngeal Abscesses.

These may be divided into acute abscesses and cold or chronic abscesses.

Their origin is as diverse as their progress. We need not dwell upon cold abscesses, which are of tubercular origin, proceed from a tubercular adenitis of the prevertebral glands or from a caries of the vertebral bodies, and thus present as a symptom of cervical Pott's disease. It will suffice to sketch briefly the main points of their evolution in the section on diagnosis.

Acute abscesses, on the contrary, by reason of their frequency and of their gravity, demand our serious attention.

Etiology and Pathogenesis.

If we review the most recent anatomical researches made in this region, we shall find that behind the pharynx, as well as behind the œsophagus, there is a sort of cavity limited posteriorly by the prevertebral aponeurosis and anteriorly by a connective-tissue sheath, designed to facilitate movements of these two organs, and on each side by a sagittal partition (Charpy) passing perpendicularly from the lamellar sheath to the prevertebral aponeurosis. This cavity, which may be distended by an insufflation of air or injection of a fluid (Escat), extends from the base of the skull to the mediastinum. At the level of the naso-pharynx it is divided longitudinally into two secondary cavities by a union of the pharyngeal aponeurosis in the middle line to the basilar apophysis and the atlas. This cavity contains within itself, in addition to the cellular tissue, but two organs on each side of the median line; these are the superior ganglion of the sympathetic, covered with a small sheath, a sort of reduplication of the sagittal septum, and Gillette's ganglion. The important vessels and nerves of the neck are situated outside of the sagittal septum, very near its external surface. If we add to these facts that Gillette's ganglion receives the lymphatics of the neck, of the naso-pharynx, and of the pharynx itself, we shall have described the region

in which retropharyngeal abscesses are developed and shall have indicated the pathogenesis of these inflammations.

As the retropharyngeal space contains only cellular tissue and lymphatic ganglia, it follows that the phlegmon can originate only in one of these two tissues. The ganglia are most frequently first attacked, but the suppuration in the cellular tissue speedily follows and then we have an adeno-phlegmon. Cellular tissue may, however, be the first to suffer; this follows when a foreign body has penetrated it, when it has been torn, when there is inflammation of the pharyngeal wall, or when the wall of the canal has been perforated by a sound, thus facilitating the introduction of septic germs; the result is an abscess which may in its turn produce secondary inflammation of the prevertebral ganglia, when we shall again find ourselves in the presence of an adeno-phlegmon.

Retropharyngeal abscesses are most commonly met with in children between the ages of six months and four or five years. Among the general causes of the affection we may mention defective nutrition and all diseases capable of causing it, such as hereditary syphilis, lymphatism, and unhygienic conditions in general, previous debility or any cachexia. These various causes weaken the organism and render it less able to contend against the pyogenic organisms.

We have often to look for the production of the disease to a buccal, pharyngeal, or nasal cause, infectious diseases being in this respect the most commonly at fault; among these we may note varicella (Stoos) which is ordinarily so benign, measles, scarlatina, erysipelas, typhoid fever, pyæmia, and urinary infection (Nélaton). A simple coryza or acute pharyngitis, an acute adenoiditis or a suppurative otitis media may produce the same result. We may understand, therefore, why retropharyngeal abscess occurs with greatest frequency in childhood, and this will be still more evident when we recall the fact that the prevertebral ganglia become atrophied in the course of time, and thus their impressionability to septic agents is weakened.

Pathological Anatomy.

We have seen above what is the location of the purulent collection. According to the part of the pharyngeal wall which is raised up by this collection of pus, we have naso-pharyngeal, buccal pharyngeal, or inferior pharyngeal abscesses. Abscesses of the naso-pharynx are the most frequent (Gillette) because they correspond directly to the seat of the prevertebral ganglia. The fact that the abscess is found on one side only may be due either to the fact that the ganglion in which the inflammation arises is located on one side or to the pres-

ence of the median septum, of which we have spoken, which acts as a barrier to the extension of the inflammatory process.

The pus, which is ordinarily fetid, due probably to the propinquity of the digestive tube, is yellow, thick, and sometimes sanguinolent. There are no specific microbes of the affection; there have been found in the pus staphylococci, streptococci, pneumococci, bacilli coli communes, and in rare cases another undescribed variety.

Symptoms.

Retropharyngeal abscess properly so-called—and we understand by that an abscess resulting from an adeno-phlegmon, and not one following the introduction of a foreign body in the post-pharyngeal tissue—is ordinarily preceded by premonitory symptoms; there may be simple coryza and sore throat or the coryza and sore throat of an eruptive fever. In adults, or at least in individuals who are old enough to take note of their sensations, the sore throat and the coryza do not pass unperceived; but in little babies the angina is often unrecognized. The coryza, in these little patients, is often betrayed by the impossibility of taking the breast; the infant seizes it with avidity, but, being unable to breathe through the nose, has to let it go almost immediately.

This is the anginous stage of authors. Then there suddenly occurs an elevation of temperature, accompanied by chills, vomiting, a coated tongue, and sometimes convulsions; the infant does not sleep, is constantly agitated, cries until it is exhausted; its cry is nasal in tone, it sometimes has attacks of coughing; it holds its head in a fixed position, the least movement imparted to it causing it to cry. Soon there occurs a more or less considerable difficulty of breathing, with intercurrent attacks of suffocation which imperil directly the life of the little patient.

Inspection of the infant's throat will enable us at once to recognize the cause of all these symptoms, for a swelling, most frequently unilateral, will have in many cases already appeared in the cervical region. It would, however, be dangerous to await the appearance of this symptom before examining the pharynx, a procedure which should never be neglected in the case of a sick child. This examination is often rendered difficult by the indocility of the little patient, or by the contraction of its jaw muscles; but as it is a question of life or death we must force an opening, using the tongue depressor, and if necessary the gag. Inspection will show us a red, purplish pharynx swelling out into the buccal cavity, the projection being more or less unilateral; this is the rule also in the beginning for abscesses occupying the naso-pharynx. In the case of an abscess of the lower portion

of the pharynx it will be well in certain cases to employ the laryngoscope notwithstanding the difficulties in the way. However, nothing can give us more clear information than the finger. At the beginning of the affection we have the diffuse doughy sensation of a soft tumor occupying the posterior wall of the pharynx. It is sometimes possible to detect fluctuation with one finger, with two fingers of the same hand, or with one of each hand curved in the form of a hook, while the physician holds the head of the child resting against his chest or abdomen, the assistants holding its hands.

The rapid evolution of the disease and the appearance of a purple, œdematous tumor in the throat will frequently enable us to dispense with palpation, which is always a painful procedure.

In addition to the symptoms which we have just mentioned, there is a rejection of liquids through the nasal fossæ, if the abscess occupy the naso-pharynx, projection forward and immobilization of the velum palati, an entrance of fluids into the respiratory passage, and a forward projection of the larynx, if the purulent collection occupy the lower portion of the pharynx.

In the absence of surgical intervention the termination will not be long delayed. The patient whose respiratory functions are more and more interfered with and who can no longer take nourishment speedily dies, for the abscess has no tendency to open spontaneously. An acute œdema of the upper portion of the larynx, combined with a pushing forward of the posterior wall of this organ, often produces asphyxia; but death may be caused in an entirely different way, namely, by infiltration of pus into the cellular tissue of the neck and into the posterior mediastinum—a rather rare complication. Bronchitis, pneumonia, or pleurisy may carry off the patient.

From the fact that the pus has no tendency to empty itself spontaneously into the pharynx we need not argue that it never does so, for it occasionally does with evil results. A bursting out of pus into the pharynx while the patient is asleep sometimes causes immediate death by asphyxia. If the opening of the abscess occur while the patient is awake it often produces only a temporary amelioration, for it does not lead to complete evacuation of the cavity, and the patient is still at the mercy of all the complications above mentioned.

Progress and Duration.

The above-described unfortunate termination of an untreated retro-pharyngeal abscess does not occur in all patients at the same period of the disease. We may, in fact, divide these abscesses into three classes, according as they have a hyperacute, an acute, or a subacute course.

The development of the first occurs with such rapidity that a slough of the pharyngeal mucous membrane may be observed within as short a period as twelve or twenty-four hours.

Those of the second category follow very nearly the course above described; the pus collects in from four to eight days, and the fatal termination takes place at any time from the fifth to the ninth day.

Subacute abscesses develop much more slowly, the retropharyngeal glandular swelling may occur in successive waves, disappearing and reappearing, and the pus may not form before the end of several weeks or even months. The local symptoms are, furthermore, much less marked and we see here a sort of transition between acute and chronic or cold abscesses. Nevertheless, the termination is just as surely fatal in the absence of surgical intervention.

Prognosis.

The gravity of the prognosis may be deduced from the above study of the evolution of the disease. It is fatal in the great majority of cases; but recovery is possible if the surgeon interfere in time.

Diagnosis.

At the beginning an erroneous diagnosis may readily be made, the physician noting only the general symptoms and having his suspicions directed toward some affection of the respiratory passages, of the digestive tube, or even of the brain.

As soon the dyspnœic and dysphagic phenomena have directed attention to the pharynx we must eliminate laryngismus stridulus, croup, eruptive fevers, and erysipelas; in very many of these diseases, indeed, there is the same suddenness of onset and the same gravity in the general symptoms, nevertheless the differences are sufficiently marked to prevent error.

Difficult dentition may sometimes be accompanied by apparently very grave general symptoms, but an inspection of the swollen alveolar processes will suffice to establish the diagnosis. It must be remembered, however, that retropharyngeal abscesses have been observed whose only cause was the eruption of a tooth, and the mere sight of a red and œdematous gum should not lead us to neglect an inspection of the pharyngeal wall.

The entrance of a foreign body into the throat is accompanied by dyspnœa, suffocative attacks, and dysphagia, which might suggest a retropharyngeal abscess; but there is no fever, and inspection and digital palpation will remove any doubts.

The presence of general symptoms, or rather their discovery, will prevent grave errors into which one might be led by inspection alone

or by a superficial palpation; we may thus escape taking for a retropharyngeal abscess a tumor of the posterior wall of the pharynx or a projection forward of the vertebral bodies in a case of cervical Pott's disease. The abscess accompanying cervical Pott's disease is of slow growth; it is painless in itself, but is accompanied by painful symptoms on the side of the vertebræ, and at the same time that there is immobility of the cervical spine there is also a displacement of the vertebræ. The abscess takes its origin behind the vertebral aponeurosis, descends along the anterior face of the bodies of the vertebræ, and seldom causes any prominence in the pharynx.

In lymphatic or tuberculous children a cold abscess may very readily take its origin from the prevertebral ganglia, occupying the same seat as a retropharyngeal abscess, and pursue a chronic course. In these cases, however, the dyspnœic symptoms appear slowly, the dysphagia increases very gradually, the abscess is almost painless to the touch, the mucous membrane preserves its normal color, and fluctuation is very marked. This kind of abscess is rare, and in any case the question of diagnosis is of minor importance, since the condition when present in young children calls for the same treatment as retropharyngeal abscess properly so called.

Treatment.

There is but one resort, namely, immediate incision. We must not wait until the pus is so abundant as to give very distinct fluctuation; as soon as the diagnosis has been established an incision should be made in the median line, parallel to the vertical axis, with a bistoury of which the point only is exposed, the rest of the blade being wrapped by a piece of adhesive plaster. The mouth gag may be used in such cases with advantage, and a few whiffs of bromide of ethyl may be given to facilitate the operation in the case of unruly children.

Recently certain authors (Watson Cheyne, Boéchat) have recommended the evacuation of the pus externally in order to assure asepsis of the sac. There can be no doubt that this method is indicated when the pus has invaded the cellular tissue of the neck and causes a projection laterally in the mastoid or submaxillary region, and we should be the first to employ the method in such a case; but to advise this procedure in all cases of retropharyngeal abscess would seem to exaggerate greatly the dangers of a direct incision, and to take too little account of the real dangers of an incision made between organs which are frequently displaced and a wound of which would be mortal.

An incision along the median line of the pharynx or in the imme-

diate neighborhood of this line presents no dangers, for the vessels (carotid and jugular) are pressed outward by the abscess and are in no danger of being wounded. After the incision the cut parts may close and a new collection of pus may then form which must be treated in the same way as the first.

We are sometimes obliged to resort to tracheotomy to avert an impending asphyxia; but that does not remove the necessity of opening the abscess, for it is only at this price that a cure can be obtained. Cases of this kind are met with especially in abscesses of the lower part of the pharynx, and to open them we often require a curved trocar or a bistoury with the blade forming an angle with the shaft.

The incision must always be made with the child's head inclined forwards, so as to avoid the passage of pus into the air tubes, and it should be followed by a thorough washing out with boric-acid solution.

The completion of the cure requires several days, but the relief of the urgent symptoms is almost immediate.

Lateropharyngeal Abscesses.

These abscesses are not peculiar to children; they originate from an acute inflammation of the tonsil or of the pharynx, from ulcerated mucous patches of the pharynx, an acute coryza, an adenoiditis, dental caries with periostitis, the cutting of a wisdom tooth, any ulceration in the pharynx, etc. We see that these causes are nearly the same as those of retropharyngeal abscess; moreover, the lateral pharyngeal abscess is sometimes merely an extension of the former, and vice versa.

Pathological Anatomy.

Here, again, we have to do with an adeno-phlegmon; it develops at the expense of the ganglia found in the maxillo-pharyngeal triangle. This triangle is limited in the inner side by the sagittal septum on which the great vascular trunks of the neck (carotid and internal jugular) rest; and the cavity is filled by the pneumogastric, the spinal accessory, the glosso-pharyngeal, and the hypoglossal nerves, a prolongation of the carotid gland, some lymphatic ganglia, and a cellulo-fatty network. If pus forms in this cavity it will press outward the great vessels and the lateral pharyngeal wall with the tonsil. These facts will give us indications for intervention.

Symptoms.

The onset is often less abrupt than in the case of retropharyngeal abscess, but it is none the less marked by alarming general symptoms,

such as chills, vomiting, trismus, delirium, and fever. Soon, at the same time with painful dysphagia and a certain degree of dyspnœa, is seen a considerable swelling of the upper lateral portion of the neck; the parotid gland is swollen, the submaxillary depression disappears and gives place to a painful bogginess, which immobilizes the head and often swells out the cervical region to a level with the masseteric and clavicular regions. The skin quickly becomes red and œdematous at the same time as the general condition grows worse; swallowing becomes more and more difficult and the respiration is embarrassed. When the contraction of the masticatory muscles is absent or but little marked, we may readily see on one side only of the throat the projecting and inflamed tonsil, the dry and inflamed pharyngeal wall surrounding the tonsil on all sides, but especially extending backwards. If we place the finger on the swelling it is easy to see that it corresponds to the projection in the lateral portion of the neck; it is even possible in a rather large number of cases to obtain by this combined internal and external palpation a sensation of fluctuation. By making a little firm pressure behind the tonsil we may sometimes feel the pulsations of the internal carotid artery, the presence of which in this place need not astonish us. Light palpation might give us the impression of carotid pulsation when none exists in this region; the cause of this error would be the systolic dilatation of the capillaries accompanying the inflammation of the pharyngeal mucous membrane.

At this stage there are a nasal intonation, difficult articulation, an abundant secretion of viscous saliva, and often a disagreeable odor of the breath, phenomena which are observed also in cases of retro-pharyngeal abscess.

Course, Duration, and Complications.

The pus in its course to the exterior travels more specially toward the angle of the jaw or toward the pharynx in a relatively short period of time (ten to fifteen days on an average after the onset of the affection). Its evacuation gives immediate relief and the wound cicatrizes with tolerable rapidity. But the termination of the case is not always so favorable. At the moment of the opening of the abscess the pus may enter the air passages and cause immediate asphyxia. But by far the most grave and the most frequent accident consists in ulceration of one of the great vessels of the neck, whence results a very grave and usually fatal hemorrhage. The blood enters the suppurating sac and forms there a false aneurysm which opens externally with the abscess. At other times the sac is already open and a fatal hemorrhage follows the penetration of the wall of the vessel as a result of the inflammatory process.

Other complications of lateropharyngeal abscess are asphyxia by compression of the air passages - a rare event—and subglottic œdema —a more frequent occurrence.

Diagnosis.

Peritonsillar phlegmon may lead to error; but in this case the pharyngeal œdema is more marked, the uvula takes the form and transparent appearance of a fishbladder, and the submaxillary swelling is less marked, sometimes even absent. The sub- and supratonsillar depression has disappeared, and the tonsil is not displaced towards the isthmus of the fauces.

Eminent surgeons have mistaken an aneurysm of the internal carotid for a lateral pharyngeal abscess; the inflammation and the absence of pulsation are not always signs which should cause the diagnosis of aneurysm to be rejected; the evolution of the tumor should be taken into serious consideration.

A superficial adeno-phlegmon of the angle of the jaw may be differentiated from lateropharyngeal abscess by the absence of swelling and of pain inside the throat; this affection is usually of dental origin.

As a lateropharyngeal abscess may occasionally travel towards the retropharyngeal space, it will be useful to determine the true point of departure of the pus. It will also be necessary to determine with care, in cases in which it is desired to open through the mouth, whether there is any pulsation between the purulent collection and the pharyngeal mucous membrane, denoting the presence of an abscess.

Prognosis.

This is grave, by reason of the possible complications; it is, however, much less serious than that of retropharyngeal abscess, for life is much less directly menaced.

Treatment.

At the beginning of the trouble we may hope, although this result is exceptional, to cause the inflammation preceding the suppuration to disappear by means of applications of Neapolitan ointment containing belladonna; this has at least the advantage of relieving the atrocious pains from which the patient suffers.

As soon as pus is formed it must be evacuated either from within or from without. We prefer the latter method, for when the abscess is opened through the pharynx there is the risk of plunging the bistoury into the carotid artery or into the jugular vein and thus, in the former case, incurring the immediate obligation to tie the common carotid.

When the incision is made through the skin, cutting the tissues carefully on a director, we may avoid many untoward accidents. Furthermore, the abscess often itself points under the angle of the jaw and thus offers an indication for a liberating incision. By means of the grooved director we may destroy the bands of fibrous tissue forming septa within the abscess, and thus favor total evacuation of the purulent collection and effective drainage. The evacuation of the pus is followed by immediate relief and the subsequent cure is rapid.

Anteropharyngeal Abscesses.

Between the posterior surface of the larynx and the anterior surface of the pharynx there is a layer of cellular tissue which occasionally becomes inflamed as a result of laryngeal disease, but abscess resulting from this inflammation is rare. When it occurs it causes an anterior projection of the pharyngeal mucous membrane and difficulty in deglutition. The laryngeal mucous membrane becomes œdematous and offers an obstacle to respiration, and even deglutition is incommoded by it.

As regards diagnosis, a gumma of this region would be with difficulty differentiated. A laryngoscopic examination will be of great service and will enable us by means of a curved bistoury to evacuate the purulent collection, and thus bring about a rapid disappearance of all the morbid symptoms dependent upon its presence.

Peripharyngeal Abscesses.

We may add, finally, that the abscess, instead of being confined to one or the other of the above-mentioned localities, may occupy the entire peripharyngeal zone, thus deserving the name of peripharyngeal abscess.

These abscesses are very grave and frequently cause the death of the patient through asphyxia and malnutrition. Their evolution is rapid and extension into the mediastinum is the rule. There is a swelling of the entire cervical region, accompanied by a very bad general condition. We have, indeed, to do with a diffuse phlegmon situated in the deeper portions of the neck, against which all surgical measures are often ineffectual.

Dry or Atrophic Pharyngitis.

Atrophic pharyngitis is a chronic inflammation of the mucous membrane of the pharynx, characterized by dryness and a certain degree of atrophy of the mucous lining of the walls of this cavity.

It has been described as a symptom of certain affections of the

pharynx, or of certain general diseases. Chomel, Morell Mackenzie, and Guéneau de Mussy have mentioned it in treating of granular pharyngitis; Isambert regarded it as the first stage of what he then (1872) called scrofulous pharyngitis, and this view was reaffirmed by Lemaître in his inaugural thesis, while Lori believed that it was an indication of an existing cardiac affection. In a word, it has received a different interpretation from each of the authors who have made it a study. Others, however, have devoted to it a special description, regarding it as an independent affection; among these we may cite Bosworth, Solis-Cohen, Fränkel, and Fayseler, and I myself am wholly in accord with their views. I believe, indeed, that typical dry pharyngitis, being the result of a special cause, deserves a special description.

Etiology.

Its etiology differs from that of ordinary pharyngitis. I leave out of consideration here the dry pharyngitis which has been observed in cases of diabetes (Joal Garel), of catarrhal inflammation of the pharynx, or of lupus, where it often constitutes the initial stage of a subsequent ulceration. The affection which will be here considered is simple atrophy of the mucous membrane of the pharynx. The causes which have been invoked for the production of dry pharyngitis are very numerous (irritating dust, vapor, etc.). I believe, however, that such an hypothesis is wide of the mark and that this form of chronic pharyngitis is of nasal origin. In atrophic coryza the nasal fossæ are enlarged as a consequence of atrophy of the turbinated bodies. There results from this a retention of the secretions of the nose, which accumulate and finally decompose. I limit myself here to a simple statement of fact, without seeking to explain for the moment the pathogenesis. Now, with these dilated nasal fossæ the current of air during both inspiration and expiration is too forcible and as a result the secretions of the pharynx become desiccated and accumulate in that region. This is, I believe, a sufficient explanation if not of dry pharyngitis, at least of a dry condition of the pharyngeal mucous membrane. But it can hardly be doubted that the atrophic process extends slowly but surely from the nasal cavities into the naso-pharynx and then into the pharynx. This is, I believe, a true explanation of the pathogenesis of real atrophy of the pharynx, an affection which is frequent in the early stages of tuberculosis, the occurrence of which it seems to favor. This nasal origin of the affection, proven at once by theory, by practice, and by clinical examination of the patient, is of manifest importance.

Among the causes has also been reckoned nasal stenosis, resulting

from hypertrophy of the turbinated bodies, from tumors, from foreign bodies, or from adenoid vegetations, but the action of such a cause is temporary, for the nasal obstruction quickly disappears. We may remark also that if there is dryness of the pharynx in such cases yet there is no atrophy of the mucous membrane; the opposite condition indeed often exists in these special forms, in which dryness is but a symptom and not the disease.

Symptoms.

Among the functional symptoms in confirmed dry pharyngitis is difficulty of deglutition, especially when the motions only are made, no bolus being actually swallowed. There is also a rather marked sensation of dryness. There is usually no cough, or if any exist it is dry, without expectoration; at times, however, there is a hoarse cough and the patient has a greenish, thick, viscous expectoration. In cases of this last class there is a general involvement of the larynx and trachea. The patient ordinarily experiences much difficulty in freeing his pharynx from the viscous mass, even sometimes crusts, which may cover the mucous membrane.

If the upper portion of the pharynx has been involved, we have also catarrh of the Eustachian tubes and more or less marked deafness. These complications are rather common.

Inspection shows an enlargement of the pharynx and of the naso-pharyngeal cavity (Solis-Cohen). The mucous membrane is dry, seeming as though covered with a layer of thick varnish, a little opaque in places and dried on the surface. The mucous membrane is somewhat wrinkled and appears thinned. In other cases there is a deposit of thick secretion of a greenish color, sometimes limited abruptly at the free border of the velum, at other times descending as far down as the entrance of the œsophagus.

On rhinoscopic examination we see that the crusts are continued into the posterior nasal fossæ, and that there exists at the same time an atrophic coryza. If we remove this secretion, which is rather difficult to do, we find a smooth, pale, colorless surface; and if we try to move the mucous membrane we shall see that it is thinner than normal and also that the muscular fibres lying behind it are atrophied.

The extension of the inflammation to the larynx has received various names; thus Luc has called it tracheal ozæna, Störck blennorrhœa; Lublinski, Michael, and H. Mackenzie have also regarded it as a special affection; but I believe that it is ordinarily merely a consequence of the condition which we are here considering, although this may not be an absolute rule, as has been shown by a case recently

(1895) communicated to the French Laryngological Society by Dr. Molinié, of Marseilles.

Diagnosis.

This is ordinarily easily made. By simple inspection we can recognize the increased size of the pharynx, the collection of secretion, and the preceding or accompanying chronic coryza. These elements will amply suffice for the diagnosis, and there need be no question of chronic pharyngitis or of dry pharyngitis from nasal stenosis. So, also, the dryness which accompanies diabetes or typhoid fever need only be mentioned to avoid confusion.

Prognosis.

In itself the prognosis of dry pharyngitis is benign, but it is aggravated by the presence of nasal or tracheal ozæna, which is often present at the same time. In patients who are obliged to use the voice professionally this complication is most serious.

Treatment.

The nasal origin of atrophic pharyngitis being admitted, we ought first to douche thoroughly the nasal and retronasal mucous membrane. These irrigations should at first be anodyne and be made with water of Salies-de-Béarn or with sulphur waters; later we may use antiseptic solutions, such as carbolic acid, chloral, resorcin, sozoiodol, etc. If the atrophic pharyngitis is very pronounced we may also prescribe sprays of carbolic acid solution or of any of the antiseptics above mentioned. Direct applications of remedies in solution are usually more efficacious than sprays. They may be made with a solution of menthol, 1:15, of chloride of zinc, 1:50 or 60, or with iodated or benzoated glycerin.

The general treatment will consist in the administration of small doses of iodide of potassium or sodium, salts which are eliminated through the salivary glands. Benzoate of sodium is also an excellent remedy in similar cases. Resort may be had to the internal use of sulphur waters, and in certain cases these may be useful as a prophylactic.

A course of treatment at some sulphurous or arsenical spring, with the various forms of local applications there practised, is often of service.

Finally, it is in this form of pharyngitis that benefit may be derived from the employment of vibratory massage (see the section on general therapeutics), the effects of which are often quite marked;

it is applied simultaneously to the mucous membrane of the nose, of the naso-pharynx, and of the pharynx. It may here awaken the vitality of the mucous membrane, favor glandular secretion, and lead, if not always to a cure, at least to a very marked amelioration.

Chronic Pharyngitis.

Varieties.—Chronic inflammation of the mucous membrane of the pharynx may be simple or granular (or glandular).

In the first form, still called exudative pharyngitis (Morell Mackenzie), the mucous membrane is smooth, of a uniform red color, often swollen at the beginning; it is generally accompanied by chronic tonsillitis and marked congestion of the velum and pillars. The dilated and sinuous vessels course through the pharyngeal wall, which is almost always covered with a limpid, grayish mucus, very adherent and analogous to that seen in the final stages of an acute pharyngitis. There are cases in which the expectoration is very abundant, and Lasègue ("Traité des Angines," p. 333, Paris, 1868) cites the case of a patient who was obliged by his profession to have long conversations in his office, who had such an abundant expectoration that he was obliged by reason of this symptom alone to give up seeing people for several weeks. According to this author the lower the part of the pharynx affected the more abundant will the secretion be. We must admit, however, that these are very exceptional cases, and it is very possible that in the case of Lasègue's patient the greater part of the liquid expectorated was made up of the saliva or of the nasal secretion.

The naso-pharynx itself is also affected and it often happens that the inflammation is localized in this part of the mucous membrane, constituting then the affection called naso-pharyngeal catarrh.

The second form, granular pharyngitis, which is much less common, has been studied in a masterly way by H. Green and N. Guéneau de Mussy.

Etiology.

We need not review the numerous causes of this affection, but will only recall the fact that it is particularly frequent among smokers,* drinkers, singers, orators, and public criers (Green, "A Treatise on Diseases of the Air Passages," New York, 1846). It has also been

*According to Ramón de la Sota (International Congress of Laryngology, Milan, 1886), there is seen in smokers sometimes a simple erythema (erythematous pharyngitis), sometimes vesicles (vesicular pharyngitis), and sometimes hypertrophied glands (granular pharyngitis).

observed in chemists, and G. Gibb ("Diseases of the Throat," London, 1864) has remarked that it is particularly noticeable in photographers and in a general way in all those who handle chemical substances in a closed apartment.

The disease is very frequent in the United States, where according to Solis-Cohen ("Diseases of the Throat and Nasal Passages," 1879) most of the chronic sore throats are characterized by inflammation of the follicular glands of the pharyngeal mucous membrane (chronic lymphadenitis). This form of throat trouble is also very frequent in France. As regards the pathogenesis of glandular angina in the different professions, we may cite the opinion of Gibb (*loc. cit.*), who believes that the greater frequency of chronic inflammation of the throat in clergymen is due in great part to the fact that they are obliged to speak in an atmosphere of which the temperature varies, often with the head bare, and exposed to draughts and currents of cold air rushing in through the open doors. In support of this opinion the author cites the case of a patient who was cured after adopting the habit of preaching with the head covered. We agree with Guéneau de Mussy in the statement that this affection is observed chiefly in men, and with Green that it is most frequent between the ages of twenty-five and thirty years, but we would also remark that glandular angina is rather frequently observed in children. In the latter it is almost always associated with hypertrophy of the tonsils and of Luschka's gland. There seems to be no difference between the sexes as regards the frequency of his form of the disease.

Symptoms.

Chronic inflammation of the pharyngeal wall is manifested ordinarily by symptoms having little relation to the extent of the lesion. Thus certain patients complaining of burning sensation, of pricking, of a constant desire to expectorate, present on a most careful examination very slight lesions, while other patients in whom inspection shows the existence of numerous, almost confluent, red granulations in the pharynx, affirm that they experience no disagreeable sensations. This fact is particularly remarkable in children, who complain but little of pharyngitis, but in whom we often discover the disease accidentally when called upon to treat them for some other affection. Moreover, the sensations experienced by the patients are of infinite variety. Ordinarily they feel a more or less sharp burning and a disagreeable tickling which causes an incessant "hem"; others have a sensation as of a pointed foreign body arrested in the throat, such as the prick of a needle, the irritation of a crust of bread, or some-

times an actual pain in the entire throat which makes the motion of swallowing painful. This last-mentioned symptom is observed especially when the granulations are seated upon the lateral walls of the pharynx.

These granulations consist in little reddish projections, more or less rounded in the form of a bean and disseminated irregularly over the wall of the buccal and nasal pharynx, sometimes even on the posterior surface of the velum and of the uvula. These projections are in typical cases surrounded by a fine vascular network, which defines very clearly their contour. The dilated and sinuous vessels are especially easy to be seen in cases in which the granulations are distinctly isolated. Sometimes we are obliged to remove with the forceps or a pledget of cotton the more or less thick or transparent gelatinous secretion covering the mucous membrane. In children the secretion is more often purulent or mucopurulent, flowing down from the naso-pharyngeal cavity over the wall of the pharynx in the form of whitish or yellowish streaks.

In certain cases inspection fails to reveal the existence of any granulations on the median wall of the pharynx and the lesion is confined wholly to the parts behind the posterior pillars. This localization, according to some authors, constitutes a special affection to which they have given the name of lateral pharyngitis. Schmidt, in 1880, was the first to give the name to this disposition of the inflamed glands (*Deutsches Archiv für klinische Medicin*, XXV., 3 and 4) and assigned to it a distinct place in the pathology of the pharynx. Two years later Dr. Heryng, of Warsaw, completed the study of this disease (*Revue de Laryngologie*, May 1st, 1882), recalling that Czermak, Störck, Michael, and Bruns had noted prior to Schmidt the existence of those elevations of the pharynx. To these names of German authors Hering might have added Grisolle, Peters, and Damaschino, who, the first in his treatise on pathology, the second in the "Dictionnaire Encyclopédique de Dechambre" (article, "Angine"), and finally the third in his treatise on "Diseases of the Digestive Tract," have each noted the existence of this interesting form of pharyngitis. In America also, Solis-Cohen ("Diseases of the Throat," 1879) says that the submucous tissue is sometimes so infiltrated beneath the lateral walls of the pharynx that there seem to be supplementary palatine folds in this region; they project, he says, along the posterior wall, extending sometimes to the pharyngeal portion of the Eustachian tube. To these observations we may add that the granulations may occupy the base of the tongue at the point of insertion of the anterior pillars—a fact which is of considerable importance in view of the trouble which these granulations may cause.

The functional symptoms are the same as those of glandular angina, but are even more pronounced, for certain patients suffer from actual neuralgia, due to the fact that at the moment of deglutition the superior constrictor of the pharynx in its contraction compresses the pad lying between it and the posterior pillars. The pharyngeal cough, the expectoration, and the voice changes have in themselves nothing characteristic. A disposition to hypochondriasis has been noted by some writers.

Lateral pharyngitis is recognized, therefore, by direct examination which shows behind each posterior pillar an oblong pad more or less granular or nodulated on its surface, the thickness of which varies from the size of a lead pencil to that of the little finger. This elevation extends as far as the orifice of the Eustachian tube and downwards as far as the aryteno-epiglottic fold, decreasing in size as it descends into the laryngeal portion of the pharynx. These projections, as we have already said, may coexist with other granulations of the pharynx or may be unaccompanied by lesions of other parts of this cavity.

According to Heryng there are two distinct forms of lateral pharyngitis: one lateral, which we have just described; the other hypertrophic, consisting in an hypertrophy of the mucous membrane which serves as a prolongation of the salpingo-pharyngeal fold. This form, in which the mucous membrane is reddish and smooth, and which may be unilateral or bilateral, is much more painful to the touch than the preceding, and the part played by the closed follicles is here purely secondary.

The diagnosis of general or lateral granular pharyngitis is too easy to require any further description here; it follows upon an inspection of the parts and is self-evident.

Prognosis.

This is grave only as regards the laryngeal and aural complications which may arise. Thus in lawyers, orators, preachers, and all who have occasion to use their voice professionally, the hoarseness which often accompanies granular pharyngitis, without being necessarily a direct consequence of it, is so troublesome as to oblige the sufferers to seek medical advice. In the same way the trouble with hearing, so common in children, calls for energetic treatment in order to avoid the deafness which might be a result of the extension of the inflammation first to the Eustachian tube and then a little later to the ear itself.

Pathological Anatomy.

Until recently very few post-mortem examinations of the structure of these pharyngeal granulations had been made and most of the his-

tological researches were upon the growths taken from the living subject. Nevertheless it was generally admitted, as the result of the researches of Sappey and Robin (quoted by Guéneau de Mussy, *loc. cit.*, p. 87) that these little elevations were due to hypertrophy of the closed follicles of the pharyngeal mucous membrane. Störck (Pitha and Billroth's "Handbuch der allgemeinen und speciellen Chirurgie," Stuttgart, 1876) alone denied the glandular character of these nodules, which he regarded as formed of swollen epithelial cells crowded against each other and deprived of their external protecting envelope.

More recently Saalfeld, quoted by Heryng (Virchow's *Archiv*, Bd. 82, 1880), in a study of this disease formulates the following conclusions: The granules and the rugosities which project above the level of the mucous membrane, attaining sometimes the size of a pea, present a gelatiniform appearance and are usually surrounded by a network of congested blood-vessels. The mucous membrane between them is but rarely hypertrophied and sometimes even atrophied. In examining these growths in the cadaver we may easily distinguish a little longitudinal cleft at the summit of each granule; this is visible especially in alcoholic preparations. Saalfeld claims to have been the first to discover this detail. Microscopic examination shows that the orifice of the excretory canal of the mucous glands is surrounded by a thick layer of swollen adenoid tissue. This layer is sometimes homogeneously infiltrated throughout the mucous tissues without forming round or oblong follicles; at other times we find in addition to this diffuse layer swollen follicles where the stroma of the reticular tissue is more condensed. The orifices of the hypertrophied glands constitute at the same time the openings of the mucous glands. The epithelium which covers these growths is more or less atrophied, so as sometimes to leave only one or two layers of cells; and it may happen that it has disappeared entirely, in which case the granulation presents on its surface a loss of substance—a fact which had already been observed by Störck.

The vessels, and especially the veins, are dilated and filled with blood; their walls, however, are normal. The red globules which distend them form a yellowish zone around the follicles, simulating an ecchymosis. These blood corpuscles are also found infiltrated in the same way in the connective tissue and the interstices of the epithelial cells; the lymphatic cells are collected in quantity around these capillaries.

Treatment.

The treatment of simple chronic pharyngitis must be directed first against the primary cause of the disease. We must, therefore,

not neglect to examine the nasal fossæ, which are very often the point of departure of the entire inflammation of the pharynx. In these cases we must direct our treatment against the lesion found. If there is an inflammation of the nasal mucous membrane it must be treated by appropriate means; if the cause resides in the abuse of tobacco or in a strain of the speaking or singing voice we must institute measures limiting, as far as possible, the action of these causes. Finally, we must endeavor to prevent acute exacerbations by means of mildly astringent gargles or sprays, such as borax, chlorate of potassium, bromide of potassium, alum, perchloride of iron, sulphate of zinc, lactic acid in dilute solution, etc. The acute attacks may be treated in the manner described above.

In the granular form, in addition to general treatment looking to a correction of the diathetic condition of the patient, local treatment will be necessary. We agree with Morell Mackenzie and Krishaber that we may obtain from local application a success which is impossible through the best devised and most scrupulously followed method of general treatment. If the burning and pricking sensations annoy the patient, compelling him to clear his throat constantly, much good may be derived from the employment of the following spray:

Antipyrin,		4.00
Hydrochlorate of cocaine,		0.25
Cherry-laurel water,		
Glycerin,	āā	0.50
Distilled water,		320.00

To this may be added 0.10 to 0.25 gm. of hydrochlorate of morphine if the disagreeable sensations still persist. I prefer the use of sprays to that of vapors in these affections. In other cases the pharyngeal wall may be painted with a solution of borax, of bromide of potassium, or of iodine in glycerin, to which may be added from one to several grams of laudanum, or 0.15 to 0.30 gm. of hydrochlorate of cocaine, to every 30 to 45 gm. of the solution. The following formula has always given me good results as a calmative of the various sensations complained of by the patient:

Iodine,	0.25
Iodide of potassium,	0.30
Sydenham's laudanum,	3.00
Tincture of guaiac,	5.00
Glycerin,	145.00

A teaspoonful of this liquid may be put into a glass of tepid water for use as a gargle or it may be applied pure on a brush to the mucous membrane of the pharynx.

Cauterization (galvanic, thermic, or chemical) should be reserved

for those cases in which the granulations cause special trouble, as when situated on the lateral walls of the pharynx or at the base of the tongue. The cauterization should be punctiform and never deep, so as to avoid producing vicious cicatrices. It should be remembered, however, that what troubles the patient is not usually the granulations but rather the local or general condition of which they are the expression. Thus we should not proceed too energetically against the follicular enlargements, but should rather seek the cause and combat or, if possible, suppress that.

For this purpose treatment at one of the various spas, with sulphur or arsenical waters, according to the case, is frequently indicated. On the other hand sea air is often too irritating and not well borne by the patient.

To repeat once more, we must not treat pharyngeal granulations simply because we see them, but only when they cause distress to the patient and when they alone are the cause of pain or discomfort.

Foreign Bodies.

The foreign bodies which are arrested in the pharynx are introduced into that cavity through the natural passages. That this arrest be possible the foreign body must be of considerable size or present irregularities on its surface. Two other factors may assist, viz., paralysis of the constrictor muscles or insensibility of the mucous membrane of the pharynx. In cases of general paralysis and in patients with multiple sclerosis these two conditions may be present. Anæsthesia alone does not appear in general to play an important rôle in the etiology of foreign bodies, for it exists in many whether hysterical or not, and the accident is not more frequent in persons with pharyngeal anæsthesia than in those with normal sensibility of this region.

The introduction of the foreign body occurs in various ways; either the patient swallows gluttonously morsels of food out of proportion to the size of the œsophagus, as often happens in the insane, general paralytics, and those suffering from multiple sclerosis; or the foreign body is swallowed out of bravado or as the result of a wager; or it is arrested in the pharynx at the moment of deglutition by reason of its shape (spicula of bone, glass, fish bones, and the like). It not infrequently happens also that objects held in the mouth are involuntarily drawn into the pharynx by a movement of deglutition and cannot then be expelled. Occasionally also living creatures, such as worms, leeches, and the like, are swallowed in water and are arrested in a fold of the mucous membrane.

Enumeration.—The principal foreign bodies found in the pharynx are: large boluses of food, pieces of bone, fruit stones, fish bones, coins, pins, needles, metallic pens, pieces of pipe stems or of glass, jewels, playthings, sets of false teeth, palatine obturators, leeches, larvæ of flies, etc.

Seat.—The lower portion of the pharynx, especially the pyriform sinuses and the region behind the arytenoid or cricoid cartilage, is the usual seat of foreign bodies. The supra-epiglottic portion of the base of the tongue, which may be considered as the antero-inferior portion of the buccal pharynx, is also a seat of predilection for sharp bodies, such as fish bones and the like.

Symptoms.

There may or may not be a history of a foreign body swallowed. The first symptom which betrays the presence of the foreign body is sometimes an attack of suffocation terminated by death. In other cases, and more frequently, the suffocative attack diminishes in violence, but there remains a desire to expectorate, a disagreeable sensation, or actual pain, increased by the slightest movement of deglutition, these being accompanied by nausea or attempts at vomiting, like those caused by the falling down, after adenotomy, of an adenoid vegetation which is still attached by a pedicle of mucous membrane. The pain has a variable seat; it is sometimes excited by pressure externally; in general, however, it is not a sign of great value. Dysphagia may or may not be present, dependent in a measure upon the shape and location of the foreign body. The respiration may or may not be impeded. In children there are frequent attacks of suffocation and a discharge of bloody mucus. In some cases after two or three hours, or perhaps two or three days, the symptoms disappear suddenly as if by enchantment, yet the original cause of them persists.

When symptoms like those above described exist the physician should institute a careful local examination. He may be guided by the index finger of the patient, which points to the location of the foreign body, but this is exceptional. Ordinarily the physician must make use of all the means at his disposal to obtain an exact appreciation of the form, the seat, and the nature of the foreign body before undertaking its removal. Inspection and palpation of the cervical and pharyngeal regions will furnish some information; digital exploration gently performed is an excellent procedure, as is also the employment of the telephone probe when we have reason to suppose that the foreign body is metallic. Better, however, than all these methods is direct inspection, obtained by depressing strongly the

base of the tongue, supplemented, if necessary, by the laryngoscope; in this way it will be scarcely possible for any foreign body to escape the eye of the observer. The œsophagoscope of Waldenburg or of Mackenzie may also be of service.

Complications.

A foreign body in the œsophagus often gives rise, either immediately or after the lapse of some time, to rather grave accidents which it will suffice merely to mention here. Thus we sometimes have emphysema, interstitial pharyngitis, or acute œdema of the vestibule of the larynx; other complications which have not infrequently been observed are immediate perforation or opening through ulceration of the large vessels of the neck, retropharyngeal abscess, caries of the laryngeal cartilages, and even caries of the vertebral bodies.

Prognosis.

The prognosis varies according to circumstances. The result may be immediate death or recovery may take place, all depending upon the seat and the shape of the foreign body, the complications, and the time of intervention.

Diagnosis.

This is ordinarily easy. In the insane, however, and in children the presence of a foreign body may not be suspected in the absence of any history pointing to such a condition; and even when it is suspected its presence may not be determined by reason of the difficulty of making a satisfactory examination of the pharyngeal cavity, especially in the insane.

A certain number of patients present themselves at the clinic complaining of the presence in the throat of a cherry stone, a pin, a fish bone, or the like which they say they feel in the pharynx and yet they are not certain of having swallowed it. Experience teaches us that these patients are usually either neurasthenics suffering from pharyngeal paræsthesia or else subjects of a slight inflammation of the follicles at the base of the tongue or of the tonsils. It will be easy to avoid error in such cases.

Treatment.

There is but one method of treatment of foreign bodies, namely, their extraction. An acute œdema or compression of the larynx sometimes necessitates an immediate tracheotomy, but that will not obviate the need of the removal of the foreign body.

Removal may be facilitated by cocaine anæsthesia and the use of the laryngeal mirror. The finger will sometimes suffice; in other cases we may be obliged to use straight or curved forceps, according to the location of the foreign body. If this has passed into the retro-pharyngeal tissue we shall be obliged to perform an internal or external pharyngotomy, according to the nature of the case, in order to avoid possible complications. In some cases it will be advantageous to push the foreign body down into the œsophagus and stomach. But we ought never to imitate the practice of former days, which was to make the patient vomit or cough or to strike him violently on the back.

Finally it must be remembered that even after the extraction of the foreign body there persists occasionally for several days or even several weeks a subjective sensation similar to that caused by its presence. It is necessary, therefore, to warn the patient, especially if he be of a nervous disposition, of the possible persistence of this sensation, in order that he may not believe that the body has been only partially extracted.

Tumors.

In this section we shall consider only tumors taking their origin in the lower portion of the pharynx, those of the naso-pharynx having been already described. Tumors of this region, as elsewhere, may be divided into benign and malignant; the sarcomas, which have generally been regarded as mixed tumors, ought to be definitely classed among the malignant growths.

Benign Tumors.

Some isolated observations of tumors of this region have been published, but there has never been any serious work published on pharyngeal tumors, the etiology of which is entirely unknown.

Pathological Anatomy.

Tumors of every variety have been met with in this region; we need only mention them, for it would be superfluous to describe in detail the histological characters of growths which occur in any part of the organism and which present nothing peculiar because of their localization in the pharynx.

Carens, Laugier, Busch, Aphavin, Taylor, and Sidney Jones have found lipomata in this region; Vibert, Ingals, MacLeod, and myself have found fibromata; Halt has seen a mucous polyp; Cozzolino a papilloma; Schuchardt and Goschler a dermoid cyst; Lennox

Browne speaks of enchondromata and osteomata which he has seen; Wolfenden and myself have observed angiomata; finally, some authors have seen aneurysm of the ascending pharyngeal or internal carotid artery develop in this region and syphilitic gummata are not uncommon. Professor Pièchaud, of Bordeaux, has observed an hydatid cyst of this region.

The seat of the tumor is variable; sometimes it arises from the posterior wall of the buccal pharynx, or from one of the lateral walls immediately below the tonsil; sometimes, on the other hand, it has its point of attachment on the anterior wall of the pharynx, on the portion of the mucous membrane which covers the posterior surface of the arytenoid region. The tonsil itself may be the point of origin of the tumor, and true adenomata—tonsillar hypertrophies long enough to pass almost into the œsophagus—have been observed. These tumors will be discussed in the article on diseases of the tonsils.

Symptoms.

In the case of certain tumors there are no symptoms. This is the rule for angiomata, the discovery of which is often purely accidental. In the early stages the presence of a tumor may be unnoticed, and the first suspicion of its existence arises only when it begins to cause functional disturbances and unusual subjective symptoms. The most common subjective signs are a sensation as of a foreign body in the pharynx, a certain difficulty of deglutition or of respiration, sometimes even of speech, and a tickling which excites coughing or vomiting. The patient, however, does not appear to suffer and preserves the aspect of good health.

In proportion as the tumor increases in size the symptoms become more and more marked, and that which was before but a simple impediment to deglutition becomes a more and more absolute obstacle to the passage of food. First solids and later even liquids pass the obstruction with difficulty and the patient despairs of being able to obtain sufficient nourishment. He emaciates but nevertheless preserves his appetite. It is impossible, however, to describe with any approach to accuracy the dysphagia which may be expected in such cases, since it will vary according to the size of the tumor, its seat, the position of the patient, etc.

Following upon a slight difficulty of respiration there may ensue with greater or less rapidity an extreme difficulty of this function. We may readily understand the reason of this if the tumor occupies the neighborhood of the larynx, which it compresses and into which it may suddenly enter causing an attack of suffocation. Hoarseness and aphonia are also due to the same causes that affect respiration.

It is evident that a tumor, even though of small size, which develops in the laryngo-pharyngeal wall will quickly cause vocal troubles of greater intensity than another tumor, even though of larger size, occupying the posterior wall of the pharynx.

The benign tumors are sessile or pedunculated; the latter may hang down into the œsophagus, occupying this tube ordinarily but sometimes passing into the mouth during an attack of vomiting. Upon direct inspection with the aid of a simple tongue depressor it will sometimes be possible to determine the presence of the tumor, its form, and its dimensions, but only when the tumor arises from the buccal pharynx. Ordinarily the laryngoscope is the only instrument by which we shall be able to acquire precise information, but this not always, for in many cases it will not be possible to see the entire tumor and especially to determine its point of implantation—a knowledge which is, however, of great importance preliminary to an operation. This is true especially in relation to tumors of the lower portion of the pharynx.

The finger is a powerful aid to diagnosis in cases where the tumor is accessible to it; it will enable us to acquire definite and useful information as to the consistence of the growth, its form, whether there is pulsation in it, etc. Sometimes the finger may be replaced by a sound having a suitable curve.

Complications.

One of the fatal complications of benign tumors of the pharynx is the sudden entrance of the neoplasm into the larynx causing immediate asphyxia; another is entrance of the tumor into the mouth during an attack of vomiting, leading sometimes to very dangerous attacks of suffocation. The epiglottis may be immobilized by the tumor so that food may fall into the larynx, an accident which is also productive of suffocative attacks.

Course, Termination, Prognosis.

The growth of the tumor is slow, but it is nevertheless progressive, and the neoplasm becomes by reason of its size more and more an obstacle to the indispensable functions of life. Even if the patient escape complications he is none the less exposed to the danger of death from hunger or of perishing from asphyxia after a longer or shorter period of time. For this reason it is often difficult to give a positive prognosis. It must be remembered, furthermore, that a benign tumor may be transformed into a malignant growth.

Diagnosis.

The first thing to be established is the existence of a tumor, and here we must remember that a projection of the vertebral bodies may be a source of error. We must then determine whether we have to do with a benign or a malignant tumor. We shall be aided in this by remembering that a benign tumor is of slow growth, is distinct from the neighboring tissues, causes no pain, is not accompanied by adenopathy, and does not affect injuriously the general condition of the patient until such time as it has acquired a volume sufficient to interfere with deglutition. In the early stages it may be difficult to establish this diagnosis, since a malignant tumor—sarcoma—may present the same characteristics. It is necessary merely to mention the possibility of confounding a tumor with retropharyngeal abscess in order to avoid falling into this error.

The differential diagnosis between a lipoma, an adenoma, a papilloma, an osteoma, and a fibroma is to be made according to the same rules that would govern the differentiation of these tumors in other regions. The diagnosis, however, would have little bearing upon the question of treatment. Pulsatile tumors should be examined with care, and we must recollect that an aneurysm may possibly give rise to no pulsations, and that mistakes have consequently arisen which have been very serious in their results to the patient.

By noting the relation of the mucous membrane to the tumor it will be possible in certain cases to say whether the latter has taken its origin from the mucous membrane, or whether, having developed in the muscular or fibrous portion of the pharyngeal wall, it has simply pushed up the mucous membrane in its growth; this information will hardly be obtained unless we can examine the pedicle of the tumor; it is, however, a matter of considerable importance from an operative point of view.

Treatment.

The only rational treatment is removal, through the natural passages if possible, or by an incision from without. If the patient can be constantly under observation we may defer operation for a number of years. There would seem to be no contra-indication to an attempt to destroy the tumor by the actual cautery or by electrolysis while it is still small. But that in itself constitutes operative interference, and it would require no longer time to remove the tumor by means of the cold wire, the galvanic loop, the écraseur, or the cutting forceps—measures which are to be recommended and which are free from the

dangers that might follow the tearing away of the growth by the forceps or fingers.

It may occasionally be necessary to resort to a preliminary operation, either to save the patient's life during an attack of suffocation, when tracheotomy will be the only resort, or to prepare the way for the removal of the tumor from without. In the latter case tracheotomy will be necessary if the tumor is of large size and closely connected with the larynx.

The special operation for the removal of the tumor is pharyngotomy, but we need not enter here into the details of the operation which is described in all works on general surgery. It goes without saying that we should beware of angiomata and that the only treatment for an aneurysm is ligation of the artery on the proximal side.

Malignant Tumors.

Malignant tumors of the pharynx may be primary or secondary, the latter being much the more frequent; they may be secondary to cancer of the larynx, of the base of the tongue, of the œsophagus, or of the tonsil.

Pathological Anatomy.

The primary tumors of this class in the pharynx comprise epitheliomata and sarcomata. The mucous membrane of the pharynx being covered with pavement epithelium, it is not surprising that we should meet here with lobulated pavement epitheliomata, which are the rule. The sarcomata may be of any variety, but the spindle-celled seem to be the most frequent.

The point of origin of malignant tumors of the pharynx is variable, but the neighborhood of the larynx is the most often invaded. We have recently had occasion to see a sarcoma attached to the lateral wall of the lower part of the pharynx but which, pushing back the epiglottis, seemed to come from the larynx.

The etiology of these tumors is obscure. One case has been reported of a solitary tumor of the posterior wall developed at the expense of the remains of the notochord.

Symptoms.

We need not dwell upon the symptomatology of these tumors, which resembles very closely that of benign tumors. As in the case of the latter, occasionally there are no symptoms in the early stages which may point to the presence of the new growth, and the tumor will have already invaded a certain portion of the pharynx before

giving rise to dysphagia, to dyspnœa, or to alterations of the voice.

The first symptom is usually hæmatemesis. If the cancer develop in the neighborhood of the larynx it will quickly immobilize the posterior part of this organ, lead to a swelling of the arytenoid mucous membrane, give rise to local and distant pains (otalgia), and will be accompanied by a rapidly increasing adenopathy about the angle of the lower jaw. The dysphagic symptoms appear especially at the time when the tumor begins to ulcerate; this is true more particularly in the case of epitheliomata, the progress of which is accompanied by a depression in the patient's general condition.

The progress of a sarcoma resembles in every respect that of a benign tumor, except as regards its very rapid growth and the evil effect upon the general health of the patient. It is accompanied by adenopathy only in the later stages, when it has begun to ulcerate.

In addition to the mechanical interference due to the presence of a malignant tumor in the lower part of the pharynx, we often observe attacks of œsophageal spasm which are much more frequent here than in the case of benign tumors. The physical signs are ordinarily sufficiently characteristic; they are the same as those accompanying cancer of the tongue. There is the same appearance of ulceration and of tubercles, the same repulsive odor of the breath, the same cancerous ichor, and the same immobilization with infiltration of the invaded parts.

The adenopathy is constant in the case of epithelioma; it is unilateral if the tumor is itself distinctly unilateral, otherwise it is found on both sides; it is manifested by an almost painless swelling of variable size of the affected glands; the latter become rapidly adherent and very hard, and project in the upper and lateral portions of the neck.

Complications.

It is not uncommon to see the adenitis take on an acute form in the course of a cancer of the pharynx, as also of the larynx, and lead to abscess of the neck. There occur more often than in benign tumors attacks of laryngeal spasm and of suffocation threatening the life of the patient.

Course and Prognosis.

The progress is ordinarily rapid and the nutrition of the patient is quickly imperilled by immobilization of the pharyngeal walls and by mechanical obstruction. The general health rapidly deteriorates and a cachectic condition ensues. The tumor does not remain limited

to the pharynx but quickly invades the larynx, the surrounding cellular tissue, and the large vessels in the vicinity. The intolerable sufferings of the patient have occasionally been terminated in a few minutes or even a few seconds through ulceration of the carotid or of the jugular vein.

Some cancers progress rather slowly here as well as in the larynx, and in their course we may see cutaneous fistulæ produced in the cervical region, and fungous growths of the same nature formed about the opening of these sinuses.

Death is the normal termination of malignant tumors of the pharynx and it occurs ordinarily quite speedily. The prognosis is therefore fatal.

Diagnosis.

We must first determine the presence of a tumor, and in this we must be guided by the rules mentioned above in the section on benign tumors. The rapid course, the early deterioration of the general health, spitting of blood, reflex otalgia, and finally the adenopathy will guide us in arriving at a correct diagnosis. The infiltration of the peripharyngeal cervical tissues occurs in an advanced stage of the affection, and as in the case of epithelioma of the larynx it will then be difficult to determine the starting-point of the tumor.

Treatment.

As a result of early interference, especially when we have to deal with a sarcoma, we may hope for complete and permanent disappearance of the tumor after ablation and cauterization of the pedicle. If the neoplasm has not invaded the neighboring parts we may still be able to prevent a return by excising widely the tissues surrounding the point of implantation. Interference should therefore be prompt, as always when we have to do with a malignant growth. The neoplasm may be removed either through the natural cavities or from without—in the latter case, after a preliminary tracheotomy, if indicated.

Sometimes tracheotomy alone will constitute an excellent palliative measure when the larynx has been invaded. The employment of the œsophageal sound, the introduction of which, however, is surrounded by some difficulties, will enable us to relieve the patient momentarily and enable him to resist for a few days longer the disease which is surely carrying him away.

DISEASES OF THE EAR.

BY

ALBERT H. BUCK,

NEW YORK CITY.

DISEASES OF THE EAR.

INTRODUCTION.

In the present treatise I shall not attempt to give more than a general sketch of the pathological changes which take place in the human ear, of the symptoms to which they give rise, of the means which are at our disposal for ascertaining the nature and extent of these changes, and of the remedial measures which are commonly employed for relieving them and for diminishing the evil effects which they may have produced. Furthermore, I shall endeavor to bear in mind, throughout this sketch, that I have been invited to write for those who are engaged in the general practice of medicine and not for men who wish to perfect themselves in the diagnosis and treatment of ear diseases. I shall therefore make no attempt to cover the entire field of aural pathology and therapeutics, but shall simply furnish such general and special information as it seems to me that the physician in active general practice ought to possess if he is to be able to advise his patients intelligently when their ears become the seat of disease.

CLASSIFICATION.

For all practical purposes it is sufficiently correct to say that the different affections of the ear may be divided into three large groups, viz., those which involve the external auditory canal, those which involve the tympanum or middle ear (by which term is meant the entire tract extending from the pharyngeal end of the Eustachian tube to the farthest outlying cells of the mastoid process), and those which involve the auditory nerve or any of its associated parts (the various cavities of what is called the labyrinth). This subdivision into diseases of the external auditory canal, middle ear, and auditory nerve is, however, correct only to this extent: it implies that a disease of one of these three parts is confined mainly to the part in question, but it does not entirely exclude the other two parts from participation in this morbid process. Indeed, as a matter of fact, all three parts of

the ear are so intimately related to one another that in the vast majority of instances it will be found that no one part is entirely exempt from the injurious influences of the disease, no matter in which of the three it may be spending its chief force. Consequently, when we speak of a disease of the middle ear, we simply mean that the morbid process, whatever may be its nature, is displaying its chief activity in some part, or perhaps in all the parts, of that complicated chain of cavities termed the middle ear.

Diseases of the External Auditory Canal.

The three principal affections to which the external auditory canal is liable are: impacted cerumen, diffuse (*i.e.*, periosteal) inflammation, and circumscribed (*i.e.*, furuncular) inflammation. Next to these, in the order of frequency, come such pathological lesions as ulcerations, granulation growths, bone caries, and the various forms of new growths.

Impacted Cerumen.

Under this term are generally included three pathological conditions, which although quite different in their etiology nevertheless present one feature in common, viz., a mass of material having all the appearances of cerumen obstructs the external auditory canal. As a matter of fact, however, these appearances are deceptive, for in only one of these conditions does the plug represent a genuine inspissation of cerumen, while in the other two it is composed either of a dried-up mass of inspissated discharge from the middle ear or of a combination of cast-off sheets of epidermis, dried pus, and cerumen—the whole representing the product of a diffuse (eczematous) inflammation of the external auditory canal. Consequently in every instance where, on first inspection, the outward appearances seem to point to a simple and uncomplicated condition of impacted cerumen, it is necessary to ascertain to which of the three classes already enumerated the case really belongs.

Impacted cerumen, pure and simple, is quite a common affection of the ears. It is encountered chiefly among comparatively young persons, and the impacted mass may present all the degrees of consistence from that of soft cheese to that of sole leather. The color also may vary from a light brown to a brownish black. Sometimes the mass is confined to the innermost end of the canal, and then again it may fill the entire space from the drum membrane to the external orifice. It is a popular idea that this condition is a very insignificant and harmless affair, to which everybody is liable and in regard

to which, therefore, one need give very little thought. Even among physicians this idea is still to a large extent the prevailing one. Taken by itself such a mass of cerumen is doubtless, under ordinary conditions of health, a perfectly harmless affair; but if the individual in whose external auditory canal this more or less hard substance is packed, happens to contract an acute inflammation of the tympanic cavity, his sufferings are likely to be materially aggravated by this complicating factor. A rapidly developing swelling of the cutaneous walls of the external auditory canal is a common occurrence in an acute inflammation of the drum cavity, and if such swelling should occur in an external auditory canal whose calibre is already more or less tightly filled with a foreign body, one may readily appreciate how greatly the sufferings of such a patient would thereby be increased.

But of even greater importance is the significance of this condition of impacted cerumen. The excessive secretion of the ceruminous glands and the accumulation of the inspissated product in the external auditory canal do not constitute an isolated and perfectly independent pathological event; they represent, I believe, a secondary or reflex manifestation of a morbid process going on in some part of the naso-pharyngeal tract. In a simple case of impacted cerumen, therefore, the cavities of the nose and the vault of the pharynx should at once be thought of as the true seat of the major disease, whatever that may be.

The other two varieties of so-called impacted cerumen possess certain characteristics which distinguish them from the genuine form which I have just described and also from each other. In simple impacted cerumen there is usually a history of sudden alternations of perfect and impaired hearing, or of the sudden development of hardness of hearing after a bath, after washing of the head, or after an attempt to clean the outer part of the canal with a towel. The same history, it is true, may be given of both the other forms, but generally in these the impairment of hearing develops more gradually. Then, again, in many cases, the history reveals the fact that more or less deafness has existed for years; this statement favoring the belief that the impaired hearing owes its origin to a sluggish or intermittent type of purulent inflammation of the middle ear, and that the plug which obstructs the canal is therefore composed largely of inspissated pus. Or the history may reveal the fact that the patient has often experienced an itching sensation in the affected ear or has noticed the occasional scaling off of the epidermis from the orifice of the auditory canal; both of which symptoms would lead us to expect that we should find a plug composed largely of epidermis, either in the form of concentric, laminated sheets or in that of a crum-

bling mass of small scales, glued together by a certain amount of cerumen and inflammatory secretion.

The latter type of plug is encountered chiefly in the middle period of life, while that which is composed largely of inspissated pus, secreted by the mucous membrane of the middle ear, may be observed at any period. The significance of the epithelial composition of the plug is not always an easy matter to determine. In the majority of cases it may be assumed that the skin of the auditory canal is the seat of an eczematous inflammation of gouty or lithæmic origin, but in some instances it is impossible to determine whether the desquamative process is an expression of such an eczematous condition or whether it may not rather be the result of a lingering periostitis of the inner half of the canal—in a word, the expression of what was once an active ostitis associated with acute inflammation of the middle ear, but which now is simply the last vestige of that earlier event.

There are no sharply drawn dividing lines between the three forms of impacted cerumen which I have here attempted to establish, and quite often there is a blending of the last two forms, sometimes even of all three.

The *relief* of the impacted condition may be effected in two or three different ways. By the aid of instruments adapted to the purpose the mass may be loosened from its attachments to the surrounding walls of the canal and then be removed, either in a single piece or in several fragments. But this mode of removal, which of course is adapted only to cases in which the impacted mass possesses a certain a degree of solidity, should be undertaken only by a person who has acquired a fair degree of skill in the use of a forehead reflector and in the management of instruments in the auditory canal. For such a person, however, this method is preferable to that which calls for the employment of a syringe and warm water. The latter very often fails, in the presence of a firm mass of cerumen, to accomplish the desired purpose within any reasonable length of time. On the other hand, when the cerumen is somewhat soft, syringing with warm water will be found to afford the best means of removing the impacted mass. But it must not be forgotten that in exceptional cases even the most carefully managed syringing of the auditory canal may give rise to a violent inflammation of the middle ear. When this happens we may be reasonably sure that the membrana tympani is perforated and that the injected stream of water, charged with particles of cerumen and all sorts of micro-organisms, has forced its way through the perforation into the middle ear. Fortunately, these cases are by no means common; but when the accident occurs the patient is very prompt in attributing it to the intervention of the doctor. I do not

know of any sure way of wholly avoiding this trouble. By employing the instrumental method of removing impacted cerumen whenever such a course is practicable, and by inquiring closely into the previous aural history of every case, we may materially diminish the number of such accidents, but now and then we are virtually compelled to use the syringe, and consequently sooner or later we shall meet with an unpleasant experience such as I have described.

The *prognosis* of these cases must always be somewhat guarded. After the removal of the impacted mass the patient may or may not experience an improvement in the hearing power. If the defectiveness of the hearing is due mainly to middle-ear lesions, the removal of the obstructing mass from the external auditory canal will aid very little or not at all in sharpening the hearing. On the other hand, if the middle ear has been fairly healthy, and if the difficulty in hearing has been due simply to the shutting out of many of the sounds of every-day life, the removal of the mass which has excluded these sounds will prove successful in restoring the hearing. On the other hand, it is a perfectly safe thing to predict that in the course of one, two, or three years the impacted condition will be reproduced.

Diffuse Inflammation of the External Auditory Canal.

This is usually a chronic disease, and is observed more often in the middle period of life than among those who are under forty. It varies greatly in intensity at different times. In some cases the only manifestation of inflammatory action is a diffuse redness of the skin lining the inner or osseous portion of the canal, and usually with this we find a similar fulness of the blood-vessels which are distributed over the handle of the hammer and around the border of the drum membrane. This hyperæmia may persist for an indefinite length of time without producing desquamation or any recognizable exudation, either upon the surface of the skin or in the interstices of its tissues. In some instances the previous history of severe middle-ear disease, often coupled with still existing lesions corroborative of such a previous attack, justifies the conclusion that the persistent redness of the skin of the canal represents merely a lingering periostitis, indicative of the fact that the underlying bone has never returned to an entirely normal condition but still remains more or less hyperæmic. These, however, are the exceptional cases. In the majority it is a fairly warranted conclusion that the chronic congestion of the skin of the canal signifies the existence of some defect in the processes of assimilation, or in those of excretion of waste material, or possibly in both. Until our knowledge in regard to such matters becomes more

complete, it is convenient to apply to this defective working of the processes of nutrition the term lithæmia or that of latent gout.

A still more active form of diffuse inflammation of the external auditory canal is that which is characterized by desquamation—sometimes in the form of branny scales of epidermis, sometimes in that of a pasty secretion which may become transformed, through drying, into scab-like masses; or the increased inflammatory activity may take the form of a more or less abundant exudation of fluid upon the free surface of the skin. In children of from one to three or four years of age this type of diffuse inflammation is often encountered as an acute affair, due to some error in diet. But it is not at all clear in these cases whether the trouble is to be considered as a reflex phenomenon, dependent upon a primary irritation of the gastro-intestinal tract, or as a true lithæmia, dependent upon the faulty materials supplied to the system for the elaboration of its blood. In any event, the trouble is of an evanescent character, disappearing spontaneously as soon as the child is put upon a suitable diet; and it should therefore not be interpreted as indicating that the little patient possesses a lithæmic or a gouty diathesis.

The *treatment* of diffuse inflammation of the external auditory canal comprises both local and general therapeutic measures. As the blood-vessels of the affected parts are in a paretic condition, one of the chief objects to be aimed at in our treatment is stimulation of their muscular elements. This may be accomplished more or less successfully by the employment of the following measures: douching with hot water (as hot as the patient can bear), local applications of nitrate of silver in solution, and the administration of strychnine or ergot internally. Another object to be attained is the exclusion of the air from the surface of the inflamed skin. For this purpose bland ointments, and preferably those which are at the same time antiseptic, may advantageously be employed. Finally, the gouty or lithæmic condition, which plays an important etiological part in so many of these cases, must be dealt with in accordance with the principles laid down in other parts of this work for the relief of this obstinate diathesis.

The *prognosis*, in this group of ear diseases, is, on the whole, favorable. In the acute variety, observed in young children, we may expect to secure complete and permanent relief. In those cases, on the other hand, where the disease is to be interpreted as a lingering remnant of what was originally a diffuse ostitis of the region involved, treatment can scarcely be expected to effect more than an amelioration of the condition. Finally, in those cases which I have classed as lithæmic, temporary cures may be looked for in a very

large percentage. In a few cases we are even warranted in pronouncing the cure to be permanent—by which I mean that for a period of several years the patient remains free from any manifestations of the old trouble. Then, again, in some of these cases of pronounced lithæmia, every therapeutic effort seems either to aggravate the evil or at least to produce no beneficial effect whatever upon its course. Such cases are observed more particularly among people who are over fifty years of age.

Circumscribed Inflammation of the External Auditory Canal; Furuncle.

In a large proportion of the cases a circumscribed or furuncular inflammation of the external auditory canal occurs in the outer half of this passage. A favorite locality is at the base of the tragus, in the lower anterior wall of the canal, close to the external orifice. In exceptional cases the furuncle may be located in the upper wall, close to the drum membrane. In this locality, however, the furuncle is almost always very small, while in the outer cartilaginous part of the canal it may easily attain such dimensions as to close the passage completely.

Pain is the most characteristic symptom of furuncular inflammation. The presence or absence of tenderness depends upon the location of the furuncle; an inflammation which is localized at or near the external orifice of the canal being accompanied by decided tenderness, while one which is situated at some distance from the orifice may reveal its existence only by spontaneous pain or by a sense of fulness in the deeper parts of the ear.

The *etiology* of furuncles in the external auditory canal is only imperfectly understood. In some instances an irritating discharge from the middle ear seems to be the exciting cause, while in others direct mechanical violence plays the same part. The diabetic diathesis is believed to favor the development of furuncles. However this may be with reference to furuncles in other parts of the body, I have never been able—by reason of the small number of cases of diabetes mellitus which have come under my observation—to verify the correctness of this belief in respect to those which develop in the external auditory canal. On the other hand, I believe that a most intimate and direct relationship exists between the lithæmic condition or diathesis and furuncles in the external ear. Indeed, so constantly are the two conditions found to coexist in the same individual that I believe I am warranted in saying that in the great majority of instances encountered in actual practice the presence of furuncles in

the external auditory canal furnishes excellent evidence that the patient is to a greater or less degree lithæmic. In many cases it will also be found that the skin of the external auditory canal is the seat of a diffuse eczematous inflammation.

In the *treatment* of the condition which we are now considering, it must not be forgotten that the furuncle is almost always a secondary production and therefore to a certain extent avoidable. The immediate cause is usually an irritating secretion that comes either from the middle ear or from the external auditory canal itself. Therefore our first care should be to secure local cleanliness—the complete removal from the auditory canal, once or twice a day, of all irritating materials. Then our next care should be to keep the skin of the canal well anointed, in order that it may be shielded from the irritating effects of any acrid secretions that may come in contact with it. The furuncle itself rarely needs to be interfered with surgically. In the exceptional cases in which such interference seems to be necessary, —cases which are characterized by intolerable pain,—it is well to wait until a sharply defined swelling reveals the exact situation of the disease and also warrants the belief that it has advanced to the stage in which a distinct sac of pus exists. The rigid, cartilage-like character of the tissues covering such a collection of pus prevents any gaping of the edges of the wound made by the knife, and consequently often diminishes greatly the degree of relief which is generally expected to follow such an incision. Hot flaxseed-meal poultices, or some form of dry heat, if the patient prefers it, may be employed as a means of mitigating the pain.

So far as the furuncles alone are concerned, a good *prognosis* may be given. They never, so far as I have observed, do serious harm, and it is only in exceptional cases that treatment fails to arrest the immediate development of other furuncles. Relapses after longer or shorter intervals are, however, by no means uncommon; and now and then a large furuncle near the orifice of the auditory canal may persist for weeks as an open pit or sinus, from which there escapes a small amount of pus.

Ulcers and Other Lesions of the External Auditory Canal.

The discussion of those affections or lesions of the external auditory canal which are of comparatively rare occurrence is scarcely suitable for a work of this character, and I may therefore be permitted to devote only a few lines to the consideration of this part of the subject.

Syphilitic ulcerations in the external auditory canal are decidedly

rare. They present no features materially different from those which characterize syphilitic ulcers in other parts of the surface of the body. Non-syphilitic ulcerations in the osseous inner half of the canal are usually to be interpreted in one of two ways: they either represent the residuum of a previous inflammation of the middle ear and neighboring bony structures, or they are the outcome of long-continued pressure exerted by some foreign substance (*e.g.*, secretions which have become inspissated and later chalky). In the former case search should always be made for a bone fistula, of which the ulcer in the canal simply represents the orifice; in the latter case, the removal of the offending mass of hard material, in combination perhaps with a little stimulation of the ulcerated surface by means of silver nitrate in crystalline form, will usually suffice to effect a cure. Active proliferation of the tissues involved in the ulceration often leads to the development of a polypoidal growth. But the presence of such a polypus in the external auditory canal does not necessarily imply that it has developed from the skin which lines that canal. In point of fact, the majority of polypoid growths observed in the outer canal are of middle-ear origin. Circumscribed bone caries is also an affection which can usually be traced back to an acute inflammation of the middle ear. Osteoma, sarcoma, and carcinoma have all been observed as independent new growths in the external auditory canal—the first quite frequently, the last two only rarely. The osteomata are perfectly harmless growths except in so far as they may, simply by reason of their size, serve to exclude sound or to interfere with the free escape of pus and other products of deeper-lying inflammation. If they are not doing harm in either of these two ways, they may be allowed to remain. The prognosis, in carcinoma of the external auditory canal, is uniformly unfavorable. So far as I know, not a single case of permanently successful removal has yet been reported. On the other hand, one of the varieties of sarcoma—viz., the osteosarcoma—seems to possess a much milder degree of malignancy, for some of them have been operated upon with what has seemed to be permanently successful results.

Diseases which Involve the Tympanum or Middle Ear.

The subdivision which is most commonly accepted is that which places the great majority of middle-ear affections in two large classes —the catarrhal and the purulent. This classification is far from correct or satisfactory, but it has been adopted for so long a period of time that it is now scarcely possible to put another in its place. And besides, no other materially better classification has yet been

brought forward. For the present, therefore, we must get along as best we may with the subdivision into *catarrhal affections of the middle ear* and *purulent affections of the middle ear.*

Those diffuse inflammations or disturbances of the tympanic mucous membrane which are characterized by swelling and congestion and by the pouring out—upon its surface and into the cavity which it encloses—of a secretion which is either watery or mucoid in character, are termed *catarrhal.* Hence the expressions *catarrhal inflammation of the middle ear, otitis media catarrhalis.* On the other hand, when the disturbance is of a more active type and the products of the inflammation assume the characters of a purulent rather than those of a serous or mucoid fluid, the terms *purulent inflammation of the middle ear* and *otitis media purulenta* are commonly employed. These different terms, therefore, simply serve to indicate two different states or conditions of the ear, but not necessarily different diseases; for to-day, in any given case, the conditions may be those of a catarrhal inflammation, while two or three days later we may find that the conditions necessitate the use of the designation purulent inflammation. *Vice versâ*, the purulent inflammation of to-day may gradually alter its character and in the course of a few days assume all the physical conditions of a catarrhal inflammation. Each of these great classes of middle-ear disease may—simply as a matter of convenience—be still further subdivided by prefixing the qualifying adjectives acute, subacute, chronic. Further on, I shall make the attempt to describe the symptoms and recognizable lesions belonging to these different types of middle-ear disease, but before doing this I think it will be well to say something with regard to the causation of diseases of the middle ear in general.

General Remarks on the Etiology of Middle-Ear Diseases.

Probably the most frequent cause of inflammation of the middle ear is *chilling of the body* when it is in a heated and perspiring condition. In a few cases the ear disturbance manifests itself very soon after the occurrence of the chilling, perhaps even sooner than does that of the nasal or pharyngeal tract. This is particularly true of those cases in which the head and neck have been exposed to a draught of cool air. Under the circumstances it is not unlikely that the middle ear is directly subjected to a chilling. But in the great majority of instances where an ear becomes inflamed through chilling of the heated body, this pathological event takes place only after the nasal or pharyngeal tract has first—and sometimes even for a period of several days—manifested all the signs of a catarrhal inflammation.

In other words, the ear affection seems to have developed, by extension, out of the nasal or pharyngeal disease, and not as one of the direct effects of the chilling of the body. We do not know what is the precise mechanism of such an extension of the disease, but it is difficult to resist the belief that certain micro-organisms play an important part in the business. The nasal cavities are at all times infested by a number of varieties of micro-organisms, some of which are known to be capable of exciting severe inflammation. So long as the parts remain healthy, these microbes seem to be powerless for evil. But when the resisting powers of the mucous membrane are lowered, as they doubtless are after the body has been chilled, it is not unlikely that the micro-organisms then cease to be harmless guests and give rise to inflammatory phenomena which vary in severity according to the number and special variety of microbes present, and also doubtless according to the degree to which the resisting powers of the mucous membrane have been lowered.

Measles, scarlet fever, small-pox, typhoid fever, and diphtheria are also prolific sources of disease of the middle ear. In these cases there can no longer be any doubt about the harmful influence exerted by the special micro-organisms belonging to each of these diseases. They penetrate into and destroy the life of the tissues which they attack; and the extent of the parts destroyed undoubtedly depends in a large measure upon the degree to which the patient's vitality has been lowered.

In the next place, inflammation may be excited in the middle ear by the accidental entrance of either salt or fresh water into that cavity by way of the Eustachian tube. During the summer months the number of cases of inflammation of the middle ear that can be traced to bathing in salt water is by no means small. In many of these cases the act of blowing the nose, at a moment when salt water is present throughout the nasal cavities, doubtless furnishes the force required for carrying the irritating fluid from the nose to the middle ear. In exceptional cases even ordinary pond or river water, which contains no appreciable amount of salt, may, when introduced into the tympanum through the Eustachian tube, excite a severe inflammation. This accident may also occur in other ways than through bathing in either salt or fresh water. It may happen, for instance, in connection with the employment of the nasal gravity douche or of the nasal syringe, and it may also follow the act of snuffing water into the nasal cavities. The effect of all these acts is to deposit a certain amount of the irritating fluid in the small pit or depression which represents the pharyngeal mouth of the Eustachian tube, from which depression the fluid is liable to be transported to the middle

ear on the very first occasion when the patient resorts to the use of the handkerchief. I have reason to believe that many physicians still think that the only danger connected with the nasal douche is that of forcing some of the fluid, as it flows *en masse* from one nasal passage into the other, up through one or the other Eustachian tube. In providing against this particular danger, therefore—which they are able to do more or less perfectly—they believe that they are doing their full duty to the patient and that they may safely continue to employ the douche even in ordinary catarrhal cases. Actual experience shows that this reasoning is not correct, and that it is better to restrict the use of the douche to the comparatively small group of aggravated cases (ozæna, syphilis) in which such a washing of the nasal passages seems clearly to be necessary.

In addition to these more quickly acting causes, there are various pathological conditions of the nasal and pharyngeal cavities which bear a very close etiological relationship to affections of the middle ear. Hypertrophy of the lymphoid tissue in the vault of the pharynx and narrowing of the nasal passages are responsible for a very large proportion of the cases of slowly developing deafness, cases which are generally placed in the category of chronic catarrhal inflammation of the middle ear. Then besides these cases of insidiously advancing deafness there are others which are characterized by repeated acute attacks of inflammation of the middle ear. This latter class includes only children and youthful individuals, and the chief if not the sole cause of these frequently recurring attacks is the presence of a mass of hypertrophied lymphoid tissue in the vault of the pharynx. The middle-ear lesions which are demonstrable in these young patients, even during the intervals of quiet, are a depressed membrana tympani, indicative of the narrowed state of the Eustachian tube, and an increased fulness of the blood-vessels, largely due, I believe, to interference with the return of venous blood to the heart by way of channels situated in close proximity to the Eustachian tube. In the other class of cases, those in which slowly increasing difficulty in hearing is the most prominent feature, the causative factor may be this same condition of hypertrophied lymphoid tissue in the pharyngeal vault, or it may be one of three or four different nasal conditions, such as a spur of bone or cartilage growing out from the surface of the septum narium, or a more diffuse thickening of this partition wall, or an enlargement of one or both of the turbinated bodies, or a polypoid outgrowth from the middle turbinated body, or an irritating discharge from Highmore's antrum into the corresponding nasal passage. It is only in comparatively recent years that some of these nasal lesions have been recognized as possessing the power to exert a harmful in-

fluence upon the organ of hearing. Exactly how this is effected we do not know precisely in every instance. Possibly reflex influences may be at work in some of the cases. In others still there may perhaps be a certain amount of direct interference with the escape of venous blood from the middle ear. And, finally, there can scarcely be any doubt with regard to the harmful effects produced upon the ear by a narrowed condition of one or both nasal passages. With every act of swallowing the air is sucked down from the nasal cavities into the throat. Under normal conditions—that is, with nasal passages freely open—the tendency to the formation of a vacuum in these cavities, and more particularly in the vault of the pharynx, must be very slight. On the other hand, if the nose is held closed during the act of swallowing, the air in the vault and in the nasal cavities will be almost completely exhausted, and the blood-vessels of the mucous membrane which is exposed to this rarefied air will become gorged with blood. Between this extreme, where the nasal orifices are closed altogether, and that which is recognized as the normal relation of these parts, all possible degrees of narrowing of the nasal passages may develop, and consequently all possible degrees of rarefaction of the air in the vault and middle ear may be produced with each act of swallowing. If this act were performed only now and then throughout the course of the day, it is not likely that the narrowed condition of the nasal passage would result in much harm to the ear. But when we consider how many times during the twenty-four hours this act of swallowing is performed, and consequently how often—I might almost say how constantly—in the condition which we are now considering, the blood-vessels of the vault and middle ear must be distended, we can form some idea of how seriously this repeated rarefaction of the air in the vault of the pharynx and communicating middle ear must affect the mechanism of hearing. But, besides those cases of impaired hearing, in which more or less stenosis of the nasal passages exists, there are others in which the septum or the middle turbinated body, or both, may be in a condition of hypertrophy and yet not encroach upon the total breathing space of the corresponding nasal channel sufficiently to produce any appreciable rarefaction effects upon the mucous membrane of the vault. In the particular cases to which I now have reference the hypertrophied turbinated body presses closely upon the opposite surface of the septum or upon the spur or ledge of bone projecting from it, and the harmful feature of this condition seems to be the pressure exerted by the two opposing mucous surfaces. It is difficult to understand how this pressure can exert any harmful influence upon the middle ear and the mechanism of hearing; and still there can be no reasonable

doubt that it does exert such an influence; for when this faulty nasal condition is relieved by surgical interference, the condition of the ear begins to improve, sometimes to a very appreciable degree. Is it possible that the harm is done through interference with the venous circulation of the Eustachian tube, or must we attribute it wholly to pathological changes brought into existence and kept active through some reflex mechanism? Until some better explanation is forthcoming I am disposed to accept the latter hypothesis as that which is most likely to be the true one. The harmful influence here referred to is also doubtless operative to a certain extent in the cases in which the condition of stenosis is the most salient feature; for the narrowing is very often, perhaps usually, associated with this same phenomenon of pressure of two opposing mucous surfaces.

In addition to the etiological factors which I have already enumerated I ought to mention gout and rheumatism. While there are no satisfactory reasons for doubting that these two diseases sometimes attack the sound-transmitting apparatus of the middle ear, as a matter of fact we do not as yet possess any data which satisfactorily establish the fact. The foot-plate of the stirrup becomes immovably fixed in the oval window, and as a result the hearing power becomes reduced to almost nothing. What is more likely than gout or rheumatism to have caused such a rigidity? If a similar immobility occurs in any other joint in the body we at once suspect one of these diseases as having been the cause of the lesion, and it seems to me that we may with equal propriety suspect these same diseases of having been the cause of a similar immobility in the stapedio-vestibular joint. But there is still another way in which one of these diseases—gout—may at least aggravate a catarrhal inflammation of the middle ear. I refer to those cases in which a latent gout gives rise to an active and generally very stubborn naso-pharyngeal catarrh, or at least intensifies an already existing catarrhal affection of this region, and so eventually involves the middle ear.

I have never been able to satisfy myself to precisely what extent, in children, diseases of the teeth, or the process called "teething," may excite an inflammation of the middle ear. I am confident, however, that the part which dental disturbances play in inflammation of the middle ear in adults must indeed be insignificant.

Methods of Examining the Middle Ear, and the Significance of the Pathological Alterations Observed.

As is well known, the tympanic cavity is very small, is situated at a considerable distance from the orifice of the external auditory

canal, and is shut off from view that is, from direct inspection—on all sides. Up to thirty or thirty-five years ago these obstacles seemed to offer an insurmountable bar to the study of diseases of the ear in the living subject. Then, thanks to the discovery of the ophthalmoscope by Helmholtz, it was found that the outer surface of the drum membrane could be well illuminated by the same sort of mirror as that which was used in illuminating the fundus of the eye, and from this time forward aural pathology made rapid advances. At the present day this department of knowledge may be said to stand on an equal footing, as regards the fulness and accuracy of the facts ascertained and also with reference to the means at our disposal for studying the phenomena of disease of the middle ear, with any other department of human pathology. The drum membrane constitutes the major part of the outer wall of the tympanic cavity, and it derives its blood supply from the same sources as does the latter. Consequently, in all processes of an inflammatory character the membrana tympani may be safely taken as an indicator of what is going on in the tympanic cavity at large. Then, in the next place, the membrana tympani is often so transparent that we can distinguish through it objects which are situated in the cavity beyond. Thus, for example, bubbles of air imprisoned in a watery or slightly mucoid fluid may often be made out without any difficulty. Then, again, the translucency of the drum membrane often permits us to recognize the fact that the mucous membrane covering the opposite inner wall of the middle ear is more abundantly supplied with blood than it should be. In such a case the light thrown into the external auditory canal passes through the membrana tympani, strikes upon the reddened mucous membrane of the inner wall of the cavity, and is then reflected back to our eyes as a diffuse reddish hue of varying intensity. Finally, the drum membrane is an air-tight diaphragm of parchment-like consistence and therefore capable of responding, to a certain extent, to whatever changes may take place in the aërostatic conditions of the middle ear. Consequently it may serve as an indicator of the degree of permeability of the Eustachian tube to the air contained in the nasal cavities and vault of the pharynx. Indeed, in this respect it is our most trustworthy guide, for nothing is easier than to be deceived in regard to the permeability of the tube when we trust, for ascertaining what its condition is in this respect, to auscultation of the ear in combination with the employment of the catheter or with any of the other methods commonly employed for forcing air through the Eustachian tube from the vault. When this passage is actually closed or even seriously narrowed for any considerable length of time—a few days, for example—the great force exerted by atmo-

spheric pressure upon the outer surface of the membrana tympani, when not counterbalanced by an equally great force pressing against its inner surface, soon pushes this membrane far beyond its natural position. Consequently, whenever we find, upon inspection with reflected light, that the drum membrane occupies a fairly natural position, we are warranted in drawing the conclusion that the Eustachian tube must be adequately pervious to air. The withdrawal of the counter-pressure follows shortly after the closure of the tube, for air then ceases to reach the middle ear, and whatever air was already in that cavity at the time when the closure took place soon becomes absorbed and its place then becomes occupied in part by serum withdrawn from the neighboring blood-vessels, while at the same time the space itself undergoes diminution in size through the advance of the membrana tympani toward the inner wall of the tympanic cavity. In another part of this chapter I shall have occasion to revert to this subject again, and I can then give further details in regard to the changes which take place in the middle ear when its channel of communication with the outer air becomes closed. For the present the foregoing statements will suffice.

There are slighter degrees of narrowing of the Eustachian tube which it is sometimes difficult to diagnose. The evidence upon which such a diagnosis is based comprises several slight alterations in the condition of the membrana tympani, both when it is in what may be termed its habitual condition and when it is in that which it presents immediately after air has been forced into the middle ear. Among these alterations may be mentioned an apparent shortening of the handle of the hammer. Under normal relations this handle seems to extend downwards as far as to the central point of the membrana tympani, but when the external atmospheric pressure is a little in excess of the internal, as happens when there is a slight degree of narrowing of the Eustachian tube, this pressure causes the hammer to rotate inward, to a greater or less degree according to the amount of the pressure exerted upon the membrana tympani. This rotation occurs about an axis which runs from before backwards and somewhat outwards through the upper limit of the drum membrane. The lower end of the handle of the hammer is lifted, by this partial rotation, into a position which is a little higher up and at the same time a little farther back than that which it occupies under normal circumstances. To the eye of the observer, therefore, the handle of the hammer will appear to have undergone a certain degree of shortening. Then, in the second place, the slight dislocation of the handle of the hammer backwards will effect another apparent change in its relation to the rest of the membrane: I refer to the fact that the angle formed

by the handle of the hammer with the posterior portion of the upper limit of the drum membrane will appear to be more acute than it is under normal conditions. A third alteration is that which takes place in the relations of the short process of the hammer. In a perfectly normal drum membrane, which is interposed between two atmospheric pressures that exactly counterbalance each other, this short process stands out very inconspicuously from the surrounding soft parts. But when the Eustachian tube becomes sufficiently narrowed to bring about a preponderance of the outer over the inner atmospheric pressure, then, under this increased pressure from without, the soft parts surrounding the short process undergo absorption, and, as the skin covering these parts recedes farther and farther inwards, the little projection of bone which represents the short process stands out more and more conspicuously.

The shortening of the "bright spot" is probably a less trustworthy indication of narrowing of the Eustachian tube than are the three alterations already enumerated above; and the same may be said of the apparent nearness of the incudo-stapedial articulation to the tympanic membrane.

Just as the drum membrane furnishes us with a trustworthy guide to what vascular changes are going on in the middle ear proper, so may we also expect the skin of the external auditory canal in the vicinity of the membrana tympani, and also at one or two other points outside of the canal, to furnish us with fairly adequate information in regard to any inflammatory changes that may be going on in the accessory cavities of the middle ear. Thus, for example, if an active inflammation is in progress in the middle ear proper, it becomes a matter of importance to ascertain early when this inflammation extends to the various cavities which are accessory to the tympanic cavity. What is called the mastoid antrum—a small cavity situated posteriorly to the tympanum and on a higher level, and communicating with the latter by a fairly broad opening—is the cavity which usually first becomes involved when the inflammation spreads. Only a comparatively thin wall of porous bone separates this cavity from the skin of the external auditory canal, at a point which lies above and somewhat behind the posterior superior periphery of the drum membrane. Furthermore, the vascular relations of this mass of bone both to the mucous membrane of the antrum and to the skin of the auditory canal are so intimate that serious inflammation of the former can scarcely take place without exciting kindred alterations in the adjacent skin of the canal. But it must not be forgotten that sclerosis of the intervening bone substance puts an end to the vascular relationship ordinarily existing in ears that have been previously

healthy, and that consequently in such cases we must not expect to see any alterations whatever in the skin of the canal, even though the antrum should be most seriously involved. There are also other routes along which an inflammation of the antrum may travel outwards and manifest itself upon the surface. These are the various emissary venous channels which traverse the body of the mastoid process, and the canal through which the facial nerve passes to open among the soft parts situated deeply on the side of the neck. Pressure upon the various spots where these channels reach the surface of the bone will be quite sure to elicit pain if the inflammation in the antrum is spreading outwards along any of these routes.

The problem of interrogating the middle ear when, through the presence of some obstacle in the external auditory canal, we are unable to obtain a view of the membrana tympani, presents at times serious difficulties. From the history of the case, however, and especially from the order in which the different symptoms have succeeded one another, a fairly correct idea can generally be obtained with regard to the nature and precise location of the disease under consideration.

Catarrhal Inflammation of the Middle Ear.

A Mildly Acute Attack.—It is only in adults or in older children that any complaint is made of the comparatively trifling disturbances caused by a mild attack of acute catarrhal inflammation of the middle ear. Occasional twinges of pain, ringing or singing or whistling sounds, a sense of pressure or fulness, very slight diminution of the hearing power, increased resonance of the patient's own voice, and perhaps a little distress caused by certain rather loud sounds that may reach the affected ear—these probably comprise all the symptoms of such a mild attack. They gradually pass away, with or without the aid of treatment, and no recognizable impairment of the hearing is left behind. In young children such mild attacks are generally overlooked, unless both ears should be involved; in which case, perhaps, the resulting deafness might be sufficiently marked to attract attention to the ears. On examination, in a case of subacute catarrhal inflammation, the drum membrane will be found to present no very marked lesions—probably nothing more than slight hyperæmia of the vessels which run along the handle of the hammer and around the upper half of the periphery of the membrane. Sometimes, instead of this picture, there will be found a little depression of the drum membrane and a slightly pinkish or purplish or even deep green reflection from the inner tympanic wall. At the same time

it will be found that some hyperæmia and infiltration of the naso-pharyngeal tract are rarely lacking; for these mild attacks are almost invariably the product of a "cold in the head."

A More Severe Attack of Catarrhal Inflammation of the Middle Ear.—In these more severe attacks the symptom of pain is usually the most striking feature, although it rarely lasts for any great length of time. If the ear is examined at this stage of the disease, the hyperæmia of the membrana tympani will be found to be more pronounced and more widespread. Not very long after the attack has begun, a blood-stained secretion is poured out into the drum cavity, often in such quantity as to cause some distention of the membrana tympani outwards. At this stage it is often impossible to determine whether we are dealing with an acute catarrhal or an acute purulent inflammation of the middle ear. But then it is not a matter of such importance that we should strive to make a differential diagnosis between the two conditions. In a number of cases which afterwards have proved to be simply catarrhal in character, I have noticed that the pain either greatly diminished in severity or even disappeared altogether when the drum cavity began to fill up with secretion. On the other hand, it is a prevailing characteristic of the purulent cases that the pain goes on increasing as the tympanic cavity becomes more and more completely filled with fluid. These two different behaviors suggest the thought that in the catarrhal variety of disease we are dealing either with a simple inflammatory process, of short-lived character, or with an inflammation started by microbes of a very mild type; whereas in the purulent variety the exciting causes of the disturbance must belong to one or more of the different species of microbes which are known to be virulent.

As a rule no permanent after-effects remain after the subsidence of one of these more decidedly acute attacks of catarrhal inflammation of the middle ear. In a few cases the absorption or dislodgment of the fluid contained in the tympanic cavity is delayed for a long time, but eventually it all disappears.

Chronic and Insidious Forms of Catarrhal Disease of the Middle Ear.—In our infirmaries, and indeed in private practice also, the cases belonging to this group are by far the most numerous. Deafness in varying degree characterizes them all, and distressing subjective noises are a frequent source of complaint. The histories of these cases are nearly all the same. First, the patient is thought for a long time to be inattentive—not to pay attention to what is said to him. Then, finally, the idea suggests itself that possibly the trouble may not be inattentiveness after all, but genuine lack of power to hear. In this way months or even years of valuable time are often

allowed to pass by before any effort is made to ascertain the cause of the loss of hearing and to apply the requisite remedial measures. I have already, in a previous section, enumerated the chief causes of these insidious catarrhal middle-ear affections, and have shown how the pictures presented by the membrana tympani are to be interpreted. There is very little to add, I believe, to what was there said. A careful and thorough examination of the nasal and pharyngeal tracts will rarely fail to reveal to us some lesion to which the damaged hearing can be primarily attributed. In some instances, these nasal or vault lesions are still potent to exert further harm upon the organ of hearing, while in others this harmful influence will be found to have ceased altogether, owing to the fact that the stage of congestion and proliferative hypertrophy, after having lasted for years, has finally given place to that of anæmia and atrophy. In a very small number of cases we fail to find any evidences warranting the belief that nasal or vault lesions have been the primary cause of the middle-ear disease, and we are then tempted to believe that a rheumatic or gouty influence has been at work diminishing the mobility of the foot-plate of the stirrup—the stapedio-vestibular articulation. In all the other articulations of the body we are able to learn something in regard to their condition by various means at our command. For example, we can interrogate their mobility and can learn whether these motions cause pain or not. Spontaneous pain in any of these joints also calls attention to it at the very beginning of a rheumatic or gouty articular affection. In the case of disease of any of the ossicular joints, however, we are debarred from all such assistance. It is scarcely conceivable that among the twinges of pain which occur from time to time in the ear, we shall ever be able to distinguish the particular kind which indicates an inflammation of one of the ossicular joints; nor can we ever hope to ascertain whether the pain elicited by passive motion of one of these joints is due to its rheumatic or gouty condition or to the roughness of our manipulations.

The *prognosis*, in these chronic catarrhal cases, is unquestionably more favorable at the present time than it was ten years ago. Very few of them at that time received any benefit from treatment, whereas now it may be claimed, with entire truthfulness, that a large percentage of this class of patients can be materially benefited by treatment. The credit for this belongs to the nasal surgeons, who have taught us that it is possible, with almost absolute safety, to operate successfully upon all those nasal and vault lesions which play the part of causal factors in so large a proportion of these so-called chronic catarrhal affections of the middle ear. The benefit consists, not merely in arresting the further progress of the secondary ear affection, but—in

a fair proportion of instances — in diminishing or entirely relieving the distressing tinnitus and in sharpening the hearing power, sometimes even to a very noticeable degree. Experience has shown that these beneficial effects often do not appear immediately after the nasal conditions have been restored to something approaching a normal state, but only after the lapse of several weeks. Long-standing paresis of the muscular elements of blood-vessels in the middle ear disappears very slowly, and this, I believe, is one of the pathological lesions which is mainly responsible for both the tinnitus and the impaired hearing.

The *treatment* of nasal affections has been discussed by another writer in an earlier article in this volume. I will reserve what I have to say upon the more directly aural treatment, for the section devoted to general aural therapeutics.

Purulent Inflammation of the Middle Ear.

The Acute Forms.

When the ear becomes inflamed in the course of an attack of measles, scarlet fever, diphtheria, or any of the diseases in which the characteristic organisms of the affection are prone to select the tympanic cavity as one of their centres of activity, we may be reasonably certain that, however mild the onset may appear to be, eventually the disturbance in the ear will assume the form of a purulent inflammation of the drum cavity. While pain is commonly a marked characteristic of such a complication, we must not overlook the fact that this note of warning is often not sounded. In young children, for example, there may be no recognizable indications of any kind pointing to the ear as the seat of serious inflammatory disturbance. Increased restlessness and a further rise in the body temperature may be the only evidences of the development of a new centre of disease. Then, again, the patient is sometimes too ill, too indifferent to sensations of all kinds to take cognizance of what may be going on in the ear. In this class of febrile diseases, therefore, it is decidedly important that the ears should be examined from time to time, in order that a free opening may be established in the membrana tympani before irreparable damage shall have been done to the organ of hearing, and before the disease shall have involved neighboring parts to a serious extent. Prolonged pressure, when the tissues pressed upon are in a low state of vitality—as they unquestionably are in all these febrile diseases—constitutes the chief danger against which we must contend. Consequently the making of such a free incision

in the membrana tympani as shall put an end to this damaging pressure is the remedial act most urgently indicated. Then, furthermore, it must be remembered that all sorts of micro-organisms, besides those which belong strictly to the particular disease from which the patient is suffering, are very likely to seize this occasion—if I may be permitted to speak figuratively of them as possessing will power—for invading the inflamed territory. We are not yet able to allot to each of these different agencies their respective shares in the damage done to the different parts of the ear. It is not unlikely though that the varieties of micro-organisms known as streptococcus, staphylococcus, and pneumococcus effect greater damage than do those organisms which belong to the fundamental disease from which the patient may happen to be suffering. Diphtheria is probably the only disease to which this statement does not rightly apply.

When the drum membrane is examined in these more serious cases, the picture presented to the eye of the observer will be found to vary greatly. Ordinarily, at the time of the examination, the inflammation will be found to have reached such a stage that one can no longer recognize any of the usual landmarks—the handle of the hammer, the well-defined periphery of the membrana tympani, etc. The inner end of the external auditory canal is occupied by a red and angry-looking surface of skin, or—if an exudation of fluid has already taken place into the inflamed tissues—the whole background will appear rather white in color, with a moist surface. But if this soaked epidermal layer be gently mopped aside at some point, the intensely red drum membrane will be found underneath. Sometimes there is no distinct bellying outwards of any part of the membrane, but more often the posterior half—that part which is nearest to the observer—will present a decidedly convex surface towards the canal.

In tubercular disease of the middle ear there is either no pain at all or else it is of a spasmodic and very mild character. The patient experiences first a sensation of fulness in the affected ear, with perhaps more or less tinnitus; then the hearing becomes impaired; and finally the ear begins to discharge. In a large proportion of the cases it is not until this stage is reached that the patient consults a physician in regard to his ear; and at this stage, unfortunately, the chief part of the damage has already been accomplished. It is doubtful, though, whether treatment is competent to materially diminish the destructive effects of this disease in the middle ear, even if the case should be seen in its very incipiency. In fact, in this early stage of the disease it is scarcely possible, by mere inspection, to determine what is the nature of the trouble—whether a

simple catarrhal attack or the beginning of an invasion of tubercle bacilli.

Syphilitic disease of the middle ear seems also to progress to the stage of breaking down of tissue and purulent discharge with but little or no pain. In private practice, however, these cases are seen much more infrequently than are those of a tubercular nature; consequently I do not feel so confident about the correctness of what I have said in regard to the painless character of this disease when it involves the middle ear.

The prognosis, in all purulent affections of the middle ear, must be guarded. In the more severe types, we can never say with certainty when or how the attack will terminate. If left to itself an acute purulent inflammation of the middle ear will, in the majority of instances, run its course without seriously threatening the life of the patient. I doubt, however, whether it can be truthfully said that it will run its course in the majority of instances without inflicting more or less serious damage upon the organ of hearing. In any event, the number of those whose hearing has been impaired or whose middle ears have been left in a state of chronic ulcerative disease through the unrestrained or only partially restrained advance of an acute purulent attack, is simply enormous. Many of these, furthermore, ultimately die from the effects of this chronic ulcerative disease, and this mortality, by fair rights, should be added to that which results directly from the acute attacks. Directly and indirectly, therefore, the mortality of purulent inflammation of the middle ear is by no means small. As recently as twenty years ago this mortality was, comparatively speaking, large, but in recent years it has been very materially reduced through the adoption of more radical surgical measures, and through the earlier recognition of danger—a step which has been rendered possible by our constantly increasing knowledge of diseases of the middle ear. The surgical measures which have proved so successful in diminishing the mortality of middle-ear diseases will be considered farther on, in the section devoted to the principles of aural therapeutics; but the consideration of the question how we may know when an acute inflammation of the middle ear is threatening danger, may fitly be taken up in the present section.

There are two fundamentally different types of serious acute inflammation of the middle ear, viz.: (1) those in which the violence of the attack is first centred in the tympanic cavity and then afterwards gradually involves adjacent parts; and (2) those in which the original inflammatory disturbance in the tympanic cavity is decidedly insignificant—too slight, in fact, to give us any special anxiety about the

ultimate issue of the disease,—while that which develops from it, in parts immediately adjacent to the tympanic cavity, shows a much greater degree of virulence. In the first of these two classes of cases we are warned in ample season of the dangerous character of the disease and are not surprised to see it spread to neighboring regions despite all our efforts to restrain it and to diminish its virulence. But in the second class, it is an easy matter for even the most experienced worker in otology to be deceived, at least for a short time, by the appearances. The external auditory canal may be dry and free from evidences of inflammatory action. The membrana tympani, although certainly not normal in appearance, may present only such alterations as are observed in the mildest types of disease. Then, in addition, there may be no redness of the skin, no swelling, no tenderness on pressure around the ear—in fact, no symptom except deep-seated and persistent pain to call our attention to the ear as a centre of disease. Fortunately, it is only in very exceptional cases that this element of pain is lacking or is very trifling in intensity. And still more fortunate is it that there is another sign which will rarely mislead us, and the appearance of which is rarely delayed for any great length of time: I refer to the drooping or prolapse of the skin covering the upper and posterior wall of the osseous external auditory canal close to the membrana tympani. It has now become recognized as a law which has very few exceptions, that serious inflammation of the mastoid antrum cannot develop without causing at the same time drooping of the posterior and upper part of the skin lining this part of the external auditory canal. While, therefore, in the majority of cases of serious acute inflammation of the middle ear there is no lack of symptoms and demonstrable lesions to warn us that danger is approaching, in even the minority we are never left wholly without a trustworthy indication of the danger at hand.

The Chronic Forms.

An acute purulent inflammation of the middle ear, of at least a moderate degree of severity, is very apt to interfere with the nutrition of some limited area of bone tissue underlying the tympanic mucous membrane, through the pressure exerted upon the latter by the fluid exudation in the tympanum. A chronic purulent inflammation of the middle ear, in perhaps a majority of instances, is due to the existence of such a limited area of bone caries, the outcome of some antecedent acute attack. Almost any part of the bony wall of the tympanum may become the seat of such a caries, but the favorite localities are the hammer and anvil, the epitympanic space, the lower edge of the entrance to the mastoid antrum, the antrum itself, etc. In a smaller

group of cases, the persistence of the discharge for months or even years after the acute attack has subsided, seems to be due, not to any lingering spot of bone caries, but to chronic irritation of the Eustachian tube and to some nasal or vault disease associated with it. The discharge in these cases is apt to be mucoid in character and perfectly odorless, and the cure of the nasal or vault disease usually puts an end permanently to the chronic discharge from the middle ear. Finally, in a very small number of these chronic cases, in which neither bone caries nor decided tubal trouble can be detected, the results of treatment seem to justify the hypothesis that the persistent but slight discharge present in these cases is due to no other lesion than a vaso-motor paresis affecting the tympanic blood-vessels.

The prognosis in any given case of chronic purulent inflammation of the middle ear depends upon several factors. If we are dealing with a case in which the nasal and tubal mucous membranes are the parts chiefly at fault, or with one in which there is no more serious lesion than a vaso-motor paresis of the tympanal blood-vessels, we may speak encouragingly of the outlook. There is no question of any danger to life, and the prospects of successful results from treatment are good. On the other hand, if bone caries is present in any part of the middle ear or communicating cavities, a more guarded prognosis must be given, both as regards the development of conditions prejudicial to life and as regards the outcome of any therapeutic procedure less radical than the so-called mastoid operation. (This expression is not to be understood in its narrow meaning, but in the broader sense of an operative attempt to remove everything that is diseased in the domain of the middle ear through an opening established in the mastoid process.) There is a decided difference between these cases of caries, both as regards the danger to life which they may involve and as regards the likelihood of our effecting at least a partial cure by the employment of either non-operative or minor operative measures. If a carious spot exists in the middle ear and the drainage from it is good, the patient may never experience any trouble from the disease beyond the slight annoyance caused by the discharge; and even this is often quite free from offensive odor. But when the drainage is imperfect—as in the case of a perforation located in the region of Shrapnell's membrane, or in that of a carious process located in an antrum the outlet of which, in the direction of the tympanic cavity, is somewhat narrowed—the effects of stagnation soon begin to show themselves and conditions are eventually established which often terminate fatally. But when these cases of stagnating and decomposing pus and other products in the mastoid antrum or neighboring cavities have not gone too far—*i.e.*, have not

already excited widespread inflammation in the vicinity—it is still often possible, partly through mechanical interference and partly through washing procedures and the employment of germicidal fluids and powders, to effect a genuine cure. In a few instances it may be found necessary, before such a cure can be effected, to remove an obstructing remnant of the membrana tympani, together with one or both of the larger ossicles—the anvil and hammer. Finally, if all these measures fail and especially if a fairly active degree of inflammation shall already have developed in the vicinity of the carious centre, the effort should be made to cut away and remove, through a large opening made in the mastoid process, and, if necessary, also to a certain extent by way of the external auditory canal, every particle of diseased bone. Theoretically, every one of these chronic cases of middle-ear disease, provided it be not of a tubercular nature, is curable. In practice, however, such faultless results are not yet uniformly obtained. At times, the explanation of this is to be found in the fact that we postpone operative interference until some vital part in the neighborhood has become involved in the inflammation. Then again in our operative work we are apt to leave some spot of carious bone behind, and to this circumstance must be attributed our failure to secure an absolute cessation of the discharge. The very great difficulty of thoroughly removing all carious bone from the vault of the tympanum, especially when the affected ear still possesses useful hearing power which we are anxious to preserve, affords some excuse for a certain percentage of these partial failures. But it is difficult to see how a valid excuse can be given when carious bone is left behind anywhere in the vicinity of the antrum or lower down in the mastoid process.

Signs which Give Warning of the Approach of Danger in Chronic Purulent Inflammation of the Middle Ear.—In the chronic cases there is usually an entire absence of certain danger signals which are commonly present in the acute cases. Thus, for example, tenderness on pressure over the mastoid process, and, later still, redness and infiltration of the skin in this region, are signs which one rarely fails to observe in the latter class of cases. Their almost uniform absence in the chronic forms of the disease is due to the fact that in these the mastoid process is almost invariably in a condition of osteosclerosis, in which condition it is almost impossible for an inflammatory disturbance located in or near the antrum to extend outwards to the surface of the bone. This osteosclerosis is the result of a slowly progressing hyperostosis which obliterates a large part of the pneumatic spaces and also most if not all of the canals through which pass the emissary blood-vessels, thus taking away the paths along which, in

the acute cases, inflammation promptly extends from the antrum to the outside mastoid integuments. Consequently in the chronic cases we must not expect to derive any light whatever upon the state of affairs in the middle ear and vicinity from the absence or presence of the three signs mentioned above, viz., tenderness on pressure, redness, and swelling of the skin behind the auricle. Less often are we deprived—through this same condition of osteosclerosis, affecting the bony floor and outer wall of the mastoid antrum—of the valuable diagnostic aid afforded by a drooping of the skin of the external auditory canal in the vicinity of the posterior and upper boundary of the membrana tympani. Pain, on the other hand, characterizes all the chronic cases in which danger is threatening. When, therefore, a case of chronic purulent inflammation of the middle ear begins to be characterized by deep-seated pain on the same side of the head, and when this pain persists despite all our efforts to relieve it, we are justified in assuming that the deep-seated inflammation, through lack of a sufficient vent, is extending dangerously near to vital parts. At this stage, if we do not postpone surgical interference for too long a time, we may feel confident of our ability to put an end to the danger. But if we wait for the appearance of still other danger signals, such as chills, rise in body-temperature, nausea or vomiting, or facial paralysis on the same side with the affected ear, our operative interference may arrive too late to save the patient.

Diseases of the Labyrinth and Auditory Nerve.

Separate and independent pathological changes undoubtedly do occur in the labyrinth, in the auditory nerve outside the labyrinth, and in the cerebral and cerebellar centres which govern this nerve; but they are of rare occurrence, and furthermore, nobody ever seems to have had the good fortune of comparing, in one and the same case, the clinical manifestations with the actual lesions as they may be seen after death. It is therefore not strange that our knowledge of diseases of the auditory nerve—both the intra- and the extra-labyrinthine portions—is extremely defective. On the other hand, the involvement of the labyrinth as a secondary phenomenon, occurring in the course of any acute or subacute inflammation of the middle ear, is an extremely frequent event. The intimate vascular relationship which exists between the tympanic cavity and that of the labyrinth will serve to explain how readily an inflammation located in the former region may spread to the latter. In the great majority of these cases the labyrinthine lesion consists of a certain degree of dilatation of its blood-vessels, and nothing more. In a certain number of instances

the profoundness of the deafness, coupled with the fact that afterwards the hearing is entirely restored, warrants the assumption that, in addition to vaso-motor paresis, a certain amount of inflammatory exudation must have been poured out into the cavities of the labyrinth, and particularly into that part which contains the nerve of hearing, viz., the cochlea. It is probably a fact that tinnitus—that is, a spontaneous ringing or whistling noise in the ear—is due to unnatural pressure upon the terminal auditory nerve filaments, and that this pressure is caused by the distention (through vaso-motor paresis) of the blood-vessels which supply the cochlea. It is clear that enlargement of the labyrinthine blood-vessels must necessarily mean increased pressure upon the contents of the labyrinth; for in the natural condition of these parts it is safe to assume that the secondary tympanic membrane is balanced to a nicety between two atmospheric pressures—the direct one upon its outer or tympanic surface, and the indirect one which is transmitted from the foot-plate of the stirrup, through a mass consisting chiefly of fluid, to its inner surface. When, however, this body of fluid is increased in bulk by the introduction into it of a larger than the normal quantity of blood—as must happen when the blood-vessels of the labyrinth are distended—the delicate balancing just mentioned ceases, all the parts are under increased tension, and—according to the degree of this tension—we shall get all the different shades of functional disturbance from a slight tinnitus to almost complete loss of hearing; for perfect freedom from tinnitus and perfectly acute hearing are scarcely possible unless this delicate balancing of the secondary tympanic membrane—or, which amounts to the same thing, of the mass of fluid filling the two scalæ and the cochleal duct—is undisturbed.

Finally, there are pathological changes in the auditory nerve—whether in the terminal apparatus in the labyrinth, or in the trunk of the nerve in the porus acusticus internus, or in the auditory nerve centres in the brain, I do not know—which must be of a more tangible and permanent character than the two which I have already enumerated. Such changes occur in the course of a number of diseases—for example, in typhoid fever, measles, scarlet fever, small-pox, diphtheria, mumps, epidemic cerebro-spinal meningitis, tuberculosis, and syphilis. Exactly what these changes are, we do not know in all cases, but we certainly cannot be far from the truth when we assume that they consist in actual rupture of blood-vessels, in the production of a new growth of connective tissue, and in infiltration of delicate structures with leucocytes and doubtless also with colonies of the different kinds of micro-organisms that are observed in these diseases. In epidemic cerebro-spinal meningitis the labyrinth is probably in-

vaded from within, that is, by way of the aquaeductus vestibuli and the porus acusticus internus; while in most of the other diseases it is quite likely that the harmful agents find an entrance from the middle ear by way of the oval or the round window. In mumps, however, it is difficult to put forward any pathological hypothesis that harmonizes with the clinical facts. When the hearing is lost, in the course of this disease, there are no associated symptoms—no recognizable middle-ear disease, no staggering gait, no nausea, no conspicuous tinnitus, no facial paralysis—nothing, in short, to throw light upon the question of how the hearing was destroyed. On the other hand, in the condition to which the term "Ménière's disease" is often applied, we have certain symptoms which justify us in assuming either that a centre of inflammation has developed in the labyrinth or, what is even more likely, that one of its blood-vessels has ruptured. There is no constant aggregation of aural phenomena in these cases of so-called "Ménière's disease," and consequently it hardly seems advisable to continue the use of a term which conveys the impression that there is a special form of ear disease to which the name may rightly be applied. The term "apoplectiform deafness," suggested by Dr. H. Knapp, of this city, is certainly preferable, for it does away with this objectionable idea of a separate and independent disease. The belief that the lesion underlying the decided vertigo and the whirling around of objects that happen to be in sight, is located in some part of the semicircular canals, is doubtless correct. It should be stated, however, that there are many authorities who believe that the injury which is unavoidably done to the brain in the course of the experiments whose object is to learn the effects of an injury done only to the contents of the semicircular canals, is the real cause of the vertigo, rotary movements, etc., and that consequently we are not warranted in accepting unreservedly the theory that the sense of equilibrium resides in the semicircular canals. Undoubtedly these authorities are in a measure right in their conclusion that an injury done to some part of the brain is competent, without any disturbance whatever of the structures in the semicircular canals, to induce the phenomena referred to above, but they are not warranted in citing this fact as proof of the incorrectness of the prevailing belief that the sense of equilibrium resides in the semicircular canals. I need only to refer to the fact that if we pull upon or injure—at any point in their course from the porus acusticus internus to the cerebellum—those filaments of the acoustic nerve which supply the ampullæ of the semicircular canals, we may legitimately expect to witness, as a result of this act, precisely the same phenomena as those which are produced by a direct destruction of the am-

pullar contents. In a word, therefore, it seems to me that the objection which they raise against the prevailing belief should be interpreted rather as confirmatory evidence in its favor. As still further proof of a clinical nature I will mention the fact that I have recently seen two cases of extensive destruction of bone tissue in the vicinity of the right middle ear in which there were good grounds for believing that the under surface of the dura mater constituted the roof of a comparatively large cavity representing the space of the recessus epitympanicus plus that which some of the neighboring bone substance had previously occupied. In both of these cases, whenever I pressed even lightly with my probe against the elastic roof of this cavity, the patient immediately experienced a decided degree of faintness and vertigo; all the objects in the room before him beginning to whirl around from left to right. In both of these cases I feel confident that no part of my probe came in contact with either the stirrup or the anvil, and consequently that the labyrinth (and more particularly the semicircular canals) was not interfered with in any manner whatever. The symptoms manifested in these two cases would seem therefore to have been produced wholly by the simple act of pushing the dura-mater, in the vicinity of the acoustic nerve, slightly upward, and thereby causing—as I infer—slight traction upon the fibres of that nerve. (I have reported these cases more fully in the Annual Report of the New York Eye and Ear Infirmary, January, 1896.)

Besides those cases of deafness which, from the lack of adequate lesions discoverable in the middle ear or external auditory canal, we feel warranted in classifying as instances of disease of the auditory nerve there are others which perhaps have a still stronger right to be thus classified. I refer to those comparatively rare cases in which the patient hears two separate and distinct tones whenever a certain note is struck upon the piano. This condition is usually of short duration and is generally observed in persons who are suffering at the time from an acute or subacute catarrhal inflammation of the middle ear. If this peculiar phenomenon be due to some disturbance in the cochlea, it must depend upon very slight and transitory lesions. Limited range of audition is another condition which is commonly believed to indicate the existence of some pathological alteration of one or the other end of the membranous lamina spiralis, with its series of superposed Corti's organs. While admitting the possibility of such cochlear lesions, I must confess that I feel more inclined to believe that the same limitation of the range of audition may be produced by some alteration in the transmitting apparatus of the middle ear.

Variously pitched tuning-forks, the piano, and Galton's whistles

supply us with the means of testing, to a certain extent, these various lesions supposed to be localized in the cochlea. But unfortunately the practical usefulness of these tests is very limited, for they can be applied successfully only to patients who possess some degree of musical cultivation. The simplest problem of this character that we are called upon to solve is that of determining, in a case of serious one-sided impairment of the hearing, whether the auditory nerve has lost its power to hear or whether we are dealing with a conducting apparatus which is no longer able to transmit sonorous impulses to the nerve. For the solution of such a question we usually employ a tuning-fork of moderately deep pitch and so clamped that the greater part of the overtones shall be excluded altogether. This fork is caused to vibrate, and, while in this state of sonorous vibration, it is placed upon the top of the patient's head, and he is asked to state in which ear he hears the tone of the fork best. If he hears it best in the ear which is not only capable of hearing well but is also free from the presence of any condition which might produce the phenomena of sound-reflection, even when the vibrating fork is placed upon the head in the immediate neighborhood of the deaf ear, we are warranted in concluding that the auditory nerve of the deaf ear is the part which is no longer able to perform its functions. On the other hand, if, as generally happens, the patient says that he hears the vibrating fork best in the deaf ear, even when it is placed upon that part of the head which is close to the sound ear, we may properly conclude that the auditory nerve is not at fault, but that some obstacle exists—either in the external canal or in the middle ear—which prevents the sound from reaching the terminal apparatus in the cochlea. The test just described is called Weber's test. Of more doubtful value is the method of testing which is commonly called "Rinne's experiment." This is based on the observation, made by Rinne in 1855, that persons with normal hearing are able to hear the sound of a vibrating tuning-fork held in front of the auricle, after they have ceased to hear the same fork by direct bony conduction through the teeth and skull. The tuning-fork is set in vibration, its handle is placed against the closed teeth or upon the head of the patient, and then, when the tone of the vibrating fork ceases to be audible to him, it is quickly transferred to a position where the still feebly vibrating prongs of the fork will be close to the orifice of the external auditory canal. If he be a person with impaired hearing, and nevertheless is able to hear the sound through the air after it has ceased to be audible through the bones of his head, the presumption is that the obstacle to good hearing is situated in the labyrinth or in some other part of the auditory nerve, and that the conducting apparatus is not affected. On the

other hand, if he is unable to perceive the sound of the fork after it has been transferred from the head to the vicinity of the outer orifice of the meatus, the presumption will be that the conducting apparatus and not the auditory nerve is at fault.

Still other methods of testing the condition of the auditory nerve have been brought forward at different times, but not one of them has so commended itself to the profession as to receive anything like general recognition.

GENERAL PRINCIPLES OF AURAL THERAPEUTICS.

In the present chapter I do not propose to lay down specific rules for the treatment of all the different pathological accidents that may befall the human ear. In the first place, as I stated in the introduction, this treatise on affections of the ear is not intended for the edification of men who are specially interested in this class of diseases, and who therefore would expect to be furnished with all the minute details of aural therapeutics, but rather for those who, although engaged in general practice, yet desire to be fairly well informed in regard to the different ways in which the ear may become diseased, the probable and possible outcome of these different affections, and the degree—as well as also, to a certain extent, the manner—in which they may be beneficially influenced by treatment. I have endeavored to carry out, in not too superficial a manner, the first two parts of the task thus outlined, and now it remains for me to complete the third. I do not feel at all sure as to what is the best manner of handling this part of my task, but I am disposed to try the plan of discussing, one after the other, some of the more important therapeutic agents and methods which are commonly employed in the treatment of diseases of the ear.

Heat and Cold.

In all those affections of the ear which are associated with pain, and more particularly in those in which the pain is the result of an inflammatory disturbance, some form of hot application rarely fails to give the patient a greater or less degree of relief. The precise form in which heat is to be utilized is not a matter of great importance. My own experience leads me to give the preference to moist heat, but there are numerous authorities who claim that they get better results from heat in a dry form. When, however, they add to this commendation of dry heat a strong condemnation of the employment of poultices in the treatment of painful inflammations of the ear, I feel disposed to protest against this judgment as unwarranted by the facts. For twenty-five years I have been in the

habit of prescribing hot flaxseed-meal poultices as a means of relieving the pain of an acute inflammation of the middle ear and also at times of a circumscribed inflammation of the external auditory canal. Not once have I been able to discover any evidences of material harm resulting from this mode of employing moist heat. In a very few instances, the skin of the auricle has been blistered by the application of too hot a poultice; but such a trifling lesion scarcely calls for even passing consideration. As to the occurrence of more serious lesions, I think it is safe to assume that carelessness and bad judgment have in some way been at the bottom of the few cases which have been reported in the medical literature of the past.

In order to enhance the effectiveness of a poultice, especially in cases of inflammation of the middle ear, it is my usual custom to have warm water (a saturated boric-acid solution, or a 1:10,000 bichloride solution, or simply water that has been boiled and then allowed to cool sufficiently) dropped into the external auditory canal until it fills this channel completely, and then immediately afterwards to have the poultice applied over the auricle and neighboring surface. The water in the canal in this case serves to conduct the heat of the poultice directly to the membrana tympani. Then, finally, as a means of prolonging the usefulness of the poultice as a source of heat, I recommend that it be covered with a blanket or something of a similar nature. In young children it is often difficult to carry out this sort of treatment. In the first place, a child's skin is decidedly more delicate than that of an adult, and consequently it is not practicable to employ any great degree of heat. Then, in the next place, it is scarcely possible to keep a child's head in one position for any length of time. To obviate the last-named difficulty it is an easy matter to fit some kind of cap to the child's head and in this way to keep the poultice in position; and as an extra protection to the sensitive skin of the auricle, the latter should be wrapped up in a piece of soft linen well anointed with vaseline, and then the poultice is to be applied over this. Effective poulticing of the ear in children demands constant attention and watching on the part of the mother or nurse; but it is particularly in children that the results are the most gratifying. As soon as it becomes reasonably certain that the pain has been diminished by the poulticing, it is a good plan to exchange some form of mild dry heat for the moist hot applications. Thus, for example, on removing the poultice we may apply over the ear and side of the head a large mass of soft cotton or wool; one or two rather loose turns of a bandage will suffice to keep the mass in place.

Now that we have learned, in recent years, how important a part micro-organisms play in inflammations of the middle ear, we can no

longer feel the same degree of confidence that we once felt in the power of heat, whether moist or dry, to check the advance of an inflammation or to moderate the force of one which has already gained considerable headway. The heat, we have reason to believe, acts as a vasomotor stimulant upon blood-vessels which are paretic—that is, unduly distended with blood; and it is possible that the vasomotor stimulation caused by the heat may not simply diminish the pain—an effect about which there can be no reasonable doubt—but may also at the same time render the mucous membrane of the middle ear a soil much less suitable for the life and activity of the micro-organisms than it previously was. If such could be shown to be the fact, we should be justified in once more adopting the belief that the heat really does possess, to a limited extent, the power to influence beneficially the course of an acute inflammation of the middle ear.

Those who advocate the employment of cold in the treatment of acute inflammation of the middle ear are not so numerous as those who prefer heat as a remedial agent, and furthermore their number seems to be diminishing. The majority of patients find the hot applications pleasanter than the cold, and, besides, the hot are rather easier to manage. Then, in some instances where the mastoid structures are involved in the inflammation, the cold seems to exercise a decided benumbing effect upon the sentient nerves, while it apparently permits the inflammation to advance in an unrestrained fashion. This difference in the therapeutic effects of heat and cold is a very important one, for the benumbing of the pain is not the effect that we particularly desire, but rather the checking or arrest of the inflammation. But the mere quieting of the pain may serve, for a considerable length of time, to conceal from us a correct knowledge of the behavior of the inflammation; for the persistence of pain affords the best kind of evidence that the disease is advancing, and consequently we cannot afford to be deprived of such valuable evidence. Hot applications, on the other hand, rarely exercise such benumbing effects, and consequently if the inflammation continues to advance, the heat will at best only partially relieve the pain, and we shall therefore not be deprived of the important knowledge furnished by the persistence of this symptom. The same sort of reasoning may be employed to show that opiates should be administered very sparingly in serious inflammation of the middle ear.

Methods of Cleansing the Cavities of the Ear.

There are various ways in which the external auditory canal can be freed from any substances that may have accumulated or become lodged there. In the case of impacted cerumen, for example, the selection of

the method to be employed will depend upon the consistence of the material to be removed. If it be quite dry and hard, the best plan is to dissect it out, either as a continuous whole or in separate fragments, by means of ring-shaped curettes with carefully rounded edges. But this can only be done by a person who is thoroughly familiar with the use of a forehead reflector and possesses a fair degree of manual skill. Those who are not accustomed to such manipulations will be compelled to employ some pattern of syringe, after doing what they can to soften the mass. This softening may often be accomplished by pouring into the affected external auditory canal a warm saturated solution of bicarbonate of soda, and allowing it to remain there for about half an hour. Then quite warm water should be injected into the canal by means of any good pattern of metal or hard-rubber ear syringe. Under the heading, Impacted Cerumen, I have endeavored to put the physician on his guard against certain accidents which may occur in the course of such an attempt to cleanse the ear. But in those cases in which the patient is obliged to cleanse his or her own ear habitually, or to have it cleansed (as in the case of a child) by some unskilled person, the problem is quite different. The piston syringe, which may be both safe and effective in the hands of a physician, is of very little use, and not entirely safe, in the hands of the patient or of the mother or nurse. Under these circumstances, the best instrument for the patient to use is the bulb syringe sold in the shops under the name of "Angelo's ear douche." The soft-rubber bulb of this instrument, which has a capacity of perhaps two ounces, is simply a reservoir that can readily be filled by first submerging it in a basin of water, then driving out all the contained air by firm pressure upon the bulb, and finally allowing it to refill itself, this time with water, through the elasticity of its own walls. A short piece of soft-rubber tubing is fastened to the bulb, and the water is sucked in through this channel. When the bulb has been filled with warm water (about 100° F. and rendered aseptic), the pronged hard-rubber nozzle should be adjusted to the free end of the rubber tubing, and the instrument will then be ready for use. The pronged nozzle is to be held lightly between the thumb and forefinger of one hand, and pushed straight into the auditory canal as far as the prongs will permit it to go. Then the bulb is grasped with the other hand and the water is injected with a fair degree of force into the auditory canal, the return current flowing out between the prongs into some vessel which is held close under the ear to catch what escapes. A glass finger bowl serves the latter purpose well. The pronged nozzle constitutes the important feature of this apparatus, for it is so constructed that the patient or the nurse can always,

without fear of doing injury to the drum membrane, push it so far into the canal that the injected stream of water can scarcely fail to play upon the membrana tympani.

Finally, there are cases in which it is necessary or desirable to cleanse the drum cavity or the neighboring mastoid antrum. This, as a matter of course, is a task which calls for considerable skill on the part of the physician. Slender glass or metal tubes, with suitably curved ends, are employed for this purpose, and air pressure is used as a means of forcing the cleansing fluid from some small reservoir (*e.g.*, a bottle with a doubly perforated cork, in which glass tubes are inserted) into a rubber tube which in turn is connected with the slender glass or metal tube.

There are some who approve of the plan of washing out the drum cavity by way of the Eustachian tube; but it does not seem to me that this method is one that should be commended.

Methods of Inflating the Middle Ear.

There are three fundamentally different ways in which the middle ear may be inflated, viz., the so-called Valsalva's experiment, Politzer's method, and that by aid of a catheter introduced through the nose into the pharyngeal orifice of the Eustachian tube.

Valsalva's experiment is so universally known that I need not describe it. It is not to be recommended for therapeutic purposes, and for various reasons. In the first place, every act of auto-inflation, as performed by Valsalva's method, is characterized by marked venous hyperæmia not only throughout the entire tract of the middle ear, but also throughout every part of the head. In the class of ear cases in which inflations are called for, one of the commonest lesions is a greater or less degree of hyperæmia due to paresis of the muscular elements of the blood-vessels. Valsalva's experiment, therefore, while it might serve, for the moment, to force the depressed drum membrane outwards towards its natural position, would in the long run only tend to aggravate one of the fundamental pathological lesions. But this is not the only objectionable feature. Experience has shown that when a patient with a depressed drum membrane is advised by his physician to resort to occasional auto-inflations (Valsalva's method), he is very apt—especially when he finds that it promptly renders his hearing more acute—to repeat the experiment many times during the day. In the course of time, as a result of this practice, the attachments of the ossicles and the ossicular articulations become so relaxed that the apparatus as a whole can no longer perform its functions in a satisfactory manner. In order to hear with any degree of comfort the patient finds it necessary to inflate his drum mem-

brane at short intervals. In a word, the ultimate result of this plan of treatment is to put the patient in a worse condition than if he had left the ear absolutely alone. It is now many years since I have seen a case such as I have described, and a fair inference from this circumstance is, that Valsalva's experiment is no longer recommended in the indiscriminate and unrestricted fashion which prevailed twenty-five years ago. For diagnostic purposes, however, there can be no reasonable objection to the occasional employment of this method.

Inflations by Means of the Eustachian Catheter.—This is the second oldest method of forcing air into the middle ear, and on the continent of Europe it has been, up to a comparatively recent date, the method most widely employed for accomplishing this particular purpose. As physicians generally are quite familiar with what is known as the Eustachian catheter, I need not describe it in this place, or state how it is to be passed through the nasal cavity into the orifice of the Eustachian tube. As I have said on various occasions, I have long since abandoned the habitual or frequent use of the catheter in the treatment of affections of the middle ear. In the first place, I feel satisfied that it is indispensable only in very rare instances. Indeed, on the spur of the moment I should not be able to state what these exceptional instances are. A cicatricial or atrophic condition of one drum membrane was at one time generally believed to exclude the employment of Politzer's method in the treatment of the other ear, or, in other words, to necessitate the employment of the catheter for the purpose of inflating that ear. But at the present time we know that the existence of an atrophic or cicatricial membrana tympani need not absolutely interfere with the employment of Politzer's inflations for the benefit of the other ear. In the great majority of these cases firm closure of the auditory canal, upon the side which corresponds with the atrophied membrane, will furnish all the needed protection against any possible injury that might result from the inflations. On the other hand, there are certain direct objections to the employment of the catheter. In the first place, its presence in the nose and in the vault of the pharynx is a decided source of irritation to these parts and indirectly to the middle ear. In the next place, patients dislike the operation greatly—much more, in fact, than they do the Politzer method of inflating the ears. Then, in the third place, I do not approve of the injection of anything but simple air into the Eustachian tube or middle ear. I am convinced from personal observation that better results can be obtained from a strict adherence to this rule of practice than from the employment of any of these remedial vapors or fluids. The less the Eustachian tube is directly interfered with therapeutically the better for both it and the patient.

Finally, there is a fourth use of the Eustachian catheter which seems to me highly objectionable. I refer to the practice of passing bougies through it into the Eustachian tube. The latter channel, it will be remembered, is encased in bone throughout a small part of its course; in this respect differing materially from the urethra. What good, may I ask, is likely to come from making pressure upon a delicate mucous membrane which rests upon an unyielding support of bone? The risk of doing actual harm is altogether too great to justify the further employment of this mode of treatment.

This brings us then to the third and most modern of the three methods of forcing air into the middle ear—the so-called *Politzer's method.* This method, which was first advocated by Prof. Adam Politzer, of Vienna, in 1861, consists in a combination of two acts performed at one and the same moment of time, viz., the compression of the air in the nasal cavities by the physician, and the simultaneous opening of the Eustachian tube by the patient; for the act of swallowing performed by the latter is also necessarily an act of opening the tube. A moment's reflection will show how greatly superior this method is to any of the other methods. In the first place, it creates no venous hyperæmia such as is called forth by Valsalva's experiment. Then, again, it sets up no irritation in the vault or nasal cavities, as happens to a greater or less degree when the Eustachian catheter is employed. Finally, it produces, I believe, a more efficient inflation, for it is the only method which permits the operator to inflate at a time when the walls of the Eustachian tube are actually pulled apart by muscular action—in other words, at the moment when the resistance to be overcome by the stream of compressed air is at its minimum. It is this characteristic which renders the regular method of procedure decidedly superior to the various modifications which have been suggested in recent years. As is well known, in the regular method the patient is instructed to take a little water into his mouth and to swallow it only when the physician gives him the proper signal. The latter, who has in the mean time inserted the end-piece of the apparatus into one of the patient's nasal openings and has then closed his fingers over the soft parts surrounding this end-piece, compresses the inflation bag one or two instants after he has given the signal to swallow. In the so-called modifications of Politzer's method the act of swallowing is omitted, and instead the patient is told to say "hic" or "hoc," or to distend his cheeks as one does in blowing a horn. Both of these acts bring the soft palate in air-tight contact with the posterior pharyngeal wall, and by thus converting the nasal passages into a closed cavity they certainly render inflation of the middle ears possible; but they do not at the same time open the

Eustachian tubes as does the act of swallowing, and consequently they are, to just this extent, less effective than the regular Politzer method of inflating the middle ears.

The beneficial effects of inflations are doubtless to be explained differently in different cases. In one, the chief gain is perhaps due to the gradual restoration of the membrana tympani to its normal position. In a second case, the benefit is to be ascribed to the pressure exerted upon the mucous membrane of the Eustachian tube and tympanic cavity; this pressure hastening the absorption of exuded materials and favoring the contraction of the blood-vessels. In a third case it is possible that some of the secretion lying free in the drum cavity may be forced out through the Eustachian tube along with the return current of air. And, finally, in still other cases it is highly probable that the sudden pushing outwards of the membrana tympani and the increased excursion imparted to the ossicles may rupture adhesions which are still so recent as to be easily torn. The beneficial results enumerated above are obtainable only in cases in which the membrana tympani is still intact. In those in which there is a perforated drum membrane, with discharge from the middle ear, no material benefit is to be expected from inflations, and it is better, in this class of cases, to abstain entirely from forcing air into the drum cavity. In certain exceptional cases, in which the mucoid character of the discharge justifies the belief that the major part of it is secreted by the walls of the Eustachian tube, we may indulge in one or two gentle inflations for the special purpose of driving the mucus entirely out of the tube, as a preparatory step to the introduction, into this diseased channel, of a suitable remedial solution (*e.g.*, a one-per-cent. silver nitrate solution). In all acute inflammations of the middle ear it is a good rule to abstain entirely from the use of inflations until the patient shall have been entirely free from pain for a period of at least two or three days. There is also another rule which it is important to bear in mind, viz., that when the condition of one ear seems to call for the employment of inflations, these must not be resorted to until the exact condition of the drum membrane of the other ear shall have been ascertained. A failure to observe this precaution may easily lead to the rupture of the unexamined membrana tympani.

Some of the Remedies Most Commonly Employed in the Treatment of Affections of the Ear.

The list of aural remedies which have received almost universal recognition is quite short. First among these stands *nitrate of silver*. This drug is employed either in the crystalline form or in that of

solutions of different strengths. When fused in the form of a small bead upon the end of a slender probe, nitrate of silver is employed for the destruction of small masses of granulation tissue in any part of the external or the middle ear, and also for the stimulation of the lining walls of a small furuncle, which, after having ruptured, remains for a long time in the condition of a pus-secreting pocket, and shows no tendency to heal. In the form of a solution silver nitrate acts chiefly as a vasomotor stimulant. The strength of the solution should vary according to both the pathological condition and the mechanical relations of the part upon which we propose to apply it. Thus, for example, if we are dealing with a diffusely inflamed external auditory canal, at the inner end of which is an intact membrana tympani, we may employ safely and advantageously almost any strength of solution. A weak solution, however, is very slow in making any impression upon such coarse tissues. Consequently, what would be termed a rather strong solution for employment in the treatment of a mucous membrane—say forty grains to the ounce, or a strength of about eight per cent.—would under these conditions be found quite mild. In cases where it is found desirable to use a silver nitrate solution in the middle ear, our decision in regard to the proper strength to employ should be influenced by the size of the perforation and by the circumstance whether we intend to force the solution through the Eustachian tube or not. Where the perforation is a mere pin-hole in size, it is best to employ a solution of such feeble concentration that little or no reaction can be expected to follow its introduction into the cavity of the middle ear. A two- or a three-grain solution (about half of one per cent.) is strong enough for such a purpose. If we especially desire to influence the Eustachian mucous membrane, we may safely force through it two or three drops of a five-grain solution (one per cent. strength). Finally, in cases which are characterized by the existence of a large perforation in the drum membrane—large enough to permit very free drainage in the event of a strong reaction from the direct effects of the silver solution—we may, with perfect safety, introduce into the middle ear solutions of almost any strength. However, there will rarely be any occasion for employing a strength greater than twenty grains to the ounce (four per cent.). The employment of nitrate of silver in almost any degree of dilution is contra-indicated in cases of tubercular disease of the middle ear.

Chromic acid and *nitric acid* are still used occasionally in aural therapeutics, although probably to a less extent than they formerly were. In cases where we desire to destroy granulation tissue, but where the location or shape of the mass will not permit us to resort to the use of the fine wire snare or to excision, pure chromic acid,

in a fluid state, furnishes us with the means of effecting this destruction safely. The spot to which this powerful caustic is to be applied should first be carefully dried, and then a very small quantity of the fluid—as much as will remain clinging to the tip end of a slender probe after it has been brought in contact with the surface only of the caustic—should be applied lightly to this spot. While nitrate of silver destroys only the tissue actually touched and shows little or no tendency to involve the deeper tissues, chromic acid, on the contrary, exerts its destructive action both laterally and in the direction of depth. It is probably safe to say that this destructive action extends to a depth of at least one millimetre. Hence the necessity for caution in using this remedy, and especially in cases where bone tissue lies at only a slight depth beneath the granulating area. Almost the only use which can be made of nitric acid in ear affections is in the treatment of the larger furuncular cavities which persistently refuse to heal. These obstinate furuncles are found on the lower wall, just inside of the orifice of the external auditory canal, and they rarely fail to heal after a single thorough swabbing of the walls of the cavity with undiluted nitric acid.

In the course of the last four or five years *peroxide of hydrogen* has won for itself a very high position as a remedy, for intratympanic use. It is specially effective in those chronic cases in which conditions of foulness and ulceration of bone exist. Its usefulness, furthermore, is not limited to its strongly germicidal powers. Besides these, it often exerts, through the effervescence which takes place when it comes in contact with decomposing organic material, a decided mechanical effect upon impacted masses of desquamated epithelium or inspissated pus, causing them to be lifted away from their attachments, and thus enabling us to dislodge them entirely from the middle ear. To this drug more than to any other is due the credit for scores of cures which nothing short of a radical operation could otherwise have effected. The solution which I ordinarily employ is one of fifteen volumes, and I never dilute it with water. So far as I have observed, no unpleasant reaction of any kind ever follows the injection of this remedy into the recesses of the middle ear. Where the perforation is small, the increased intratympanic pressure produced by the effervescence may cause a little vertigo, but it passes off in the course of a few moments. The injections should be repeated two or three times in succession, at short intervals or until the injection of a fresh quantity of the remedy no longer gives rise to a noticeable degree of effervescence.

Of the other solutions or fluids which are occasionally used in intratympanic medication, it is sufficient simply to enumerate the

following three, on account of some slight merit which they possess under certain circumstances: a saturated solution of *boric acid,* a weak (1:10,000) *bichloride of mercury* solution, and *absolute alcohol.*

Among the powders there are several which have been found useful in the treatment of chronic purulent middle-ear disease. These are iodoform, dermatol, aristol, boric acid, and burned alum. After the cavity of the tympanum has been cleansed as thoroughly as possible by the injection of peroxide of hydrogen, it will be found desirable to introduce some remedy in powder form which shall perpetuate, at least for a day or two, the aseptic condition effected by the cleansing. *Iodoform* may be trusted to accomplish this purpose more surely than any of the other powders which I have enumerated. Unfortunately, some patients object so seriously to its penetrating odor that we find ourselves compelled to abandon its use. *Dermatol* has scarcely any odor and is perhaps sufficiently effective as a germicide, but it rapidly becomes converted from the condition of a powder to that of a very adhesive paste. This characteristic renders it decidedly inferior to iodoform, which seems to retain its powdery form for a long time. Finely powdered *boric acid* is sometimes objectionable on account of its tendency to form hard, chalk-like masses in the presence of a little moisture. For this reason it should be introduced more sparingly into the tympanic cavity, or into a comparatively narrow sinus. *Aristol* seems to be the least desirable of the antiseptic powders that are employed in the treatment of chronic purulent disease of the middle ear. Occasionally it seems to exert a slightly irritant action upon the tissues, and furthermore it does not appear to possess as strong germicidal powers as do the other powders enumerated. *Burned alum* is used in chronic middle-ear affections simply on account of its astringent powers. It should be applied in very small quantity, and care should be taken not to drop any of it on the floor of the external auditory canal, as it seems to possess the power of setting up furuncular inflammation in the skin of this passage. The practice of blowing any of the powders above enumerated indiscriminately into the deeper portions of the external auditory canal is not to be commended. By means of a slender probe, suitably bent, it is possible, under proper illumination with the mirror, to lodge our antiseptic powder in precisely the locality where we desire it to be deposited and in such quantity as may seem to us desirable. When the openings are sufficiently large, we may, by wrapping a little cotton around the bent end of the probe, be able to introduce quite large instalments of powder into the recesses of the midde ear.

Operative Procedures in the Treatment of Ear Diseases.

Among the operations which have been performed upon the ear there are comparatively few which have stood the test of time and are now generally admitted to be valuable additions to our resources for relieving aural disease. First among these should be mentioned *paracentesis of the membrana tympani*. The value of this operation varies greatly according to the purpose for which it is performed. When the incision is made for exploratory purposes—as, for example, to enable the operator to ascertain what are the contents of the tympanic cavity or what is the condition of the ossicular articulations—its value is comparatively small. Paracentesis is also performed for the purpose of simply emptying the tympanic cavity of its liquid contents, as for example in cases of what was formerly termed otitis media serosa or mucosa—cases in which there is no question whatever of increased intratympanic pressure. The operation, however, is not performed for this purpose as often as it was twenty years ago, for it has been found that the ultimate return of the tympanic cavity to its normal condition is not materially hastened by such interference. On the other hand, as a means of putting an end to a degree of intratympanic pressure which threatens to destroy the life of some part or parts of the mucous membrane pressed upon, and, further, as a means of furnishing an easy and direct drainage channel for all sorts of pathological products that would otherwise remain imprisoned in the various chambers and recesses of the middle ear, paracentesis of the membrana tympani is appreciated at as high a value to-day as it was more than a score of years ago. It is no exaggeration to say of it that it has saved hundreds of lives and that it has relieved thousands from the misery of being left with more or less decided impairment of the hearing or with a chronic discharge from the ear. I do not propose to describe this operation, but I wish to lay stress upon the fact that, in the hands of any reasonably careful and reasonably skilful person, it is entirely free from danger. The knife commonly employed for making the paracentesis is a miniature spear, and the part of the membrane which is usually selected for the cut is the posterior half, which is nearer to the operator's eye and which contains but one small area where the penetrating point of the knife might do some harm. If we divide the membrane, for purposes of description, into four nearly symmetrical areas or quadrants, using the handle of the hammer as the dividing-line between the two upper quadrants, and then if we again subdivide the posterior upper quadrant into halves—an upper and a lower half—the dangerous area referred to above will be

found to lie wholly within the limits of the upper one of these two halves. In this dangerous area are to be found, directly behind the intervening membrana tympani, the incudo-stapedial joint and the stirrup itself. A careless or clumsy manipulation of the knife in this dangerous area would be very likely to seriously damage the stirrup and perhaps even to destroy the hearing of that ear. But below this area there is not a single structure of any importance that might receive harm from the point of the knife. The latter may enter the niche for the fenestra rotunda but it cannot reach or injure the secondary tympanic membrane. It is therefore perfectly safe to introduce the point of the knife into the drum membrane just below the dangerous area described above and then to carry the incision in a curving direction downwards and forwards until it extends well into the anterior lower quadrant. At this point it is again necessary to exercise a certain degree of caution, not for the sake of avoiding some important structure—for there are no important structures in the neighborhood of this quadrant—but simply because the mucous membrane of the inner wall of the tympanum begins at this point to be very rich in blood-vessels, and consequently too deep an incision would here be likely to be followed by the formation of a blood-clot in the middle ear. This accident, which of itself possesses scarcely any importance, may easily rob the patient of nearly all the benefits that may legitimately be expected to flow from the paracentesis; the clot of blood effectually sealing, sometimes for hours together, the crescentic opening made by the knife.

There are a number of operations upon the ossicles and membrana tympani which have, each in turn, had enthusiastic adherents, but which to-day belong rather to the history of otology than to the list of remedial measures commonly employed in otological practice. Partial or complete excision of the membrana tympani for the relief of deafness; division of the tendon of the tensor tympani muscle or division of the posterior fold of the drum membrane for the relief of distressing tinnitus; excision of the hammer and anvil for the relief of the same symptom and also at times for the purpose of improving the hearing; finally, the removal of the entire stirrup (stapedectomy), for the purpose chiefly of revivifying the hearing power—these are all operations which seem destined to be abandoned by conservative men. On the other hand, ossiculectomy—by which term is usually meant the excision of only two of the ossicles, viz., the hammer and the anvil—is to be commended as a very useful operation in certain cases of bone caries involving the epitympanic space or the mastoid antrum. In this statement I have limited the field of usefulness of the operation simply because, as I stated on a previous page, in a

large number of these cases the conditions are such that a cure can be effected by simple cleansing and antiseptic procedures.

Then, finally, we have a group of operations which in all essential respects are one operation and may therefore be conveniently described as the *mastoid operation*. They all have one object in common, viz., to remove from the temporal bone all diseased tissues and all septic or irritating products of disease which may have become lodged in its cavities or recesses; and in the accomplishment of this object they all, at the beginning, follow the same route, viz., through the skin covering the mastoid portion of the temporal bone and then through the bone itself until the mastoid antrum is reached. Then after this part of the operator's task has been accomplished he may find it necessary—in order to comply with the rule that all diseased tissue and all irritating or septic products of disease must be removed—to lay bare the dura mater above the tympanum or the antrum, to cut away the bone substance lying between the latter cavity and the sigmoid flexure of the lateral sinus, in a word, to destroy a large part of the external portions of the temporal bone. In this work he must constantly keep well in mind the anatomical relations of all these parts, for injury to the stirrup may mean complete destruction of the hearing, and tearing of the facial nerve where it passes horizontally over the fenestra ovalis, or in its downward course in the vicinity of the lower margin of the entrance to the mastoid antrum, may leave the patient with a disfiguring facial paralysis. I should add, however, that the areas of bone caries may be situated so close to the stirrup and the oval window that an injury to either the stirrup or the facial nerve, or to both, can scarcely be avoided. In other words, neither anatomical knowledge nor the exercise of great care and skill affords a sure protection against the two accidents mentioned. The danger of inflicting a serious injury upon either the dura mater or upon the wall of the lateral sinus is comparatively small when reasonable care is exercised in the management of chisels, scoops, etc.

The prognosis, in mastoid operations, is decidedly favorable. If the histories of the fatal cases be carefully analyzed, it will be found, I am quite confident, that with rare exceptions the fatal issue is to be ascribed to one of two things: either that the operation has been performed at too late a date, that is, after the ear affection has already given rise to serious intracranial disease; or else that the operative interference has been carried out too timidly, and as a consequence has not removed all diseased tissue and all the septic products of the disease. Exact statistics relating to the operation in question cannot be given, but I believe that at the present time the successful cases

represent at least eighty per cent. of the total number. In course of time, when comparatively few cases will be permitted to reach such an advanced stage of disease before the operation is performed, I have no doubt that the mortality will be reduced to ten or even five per cent.

DISEASES OF THE TONSILS.

BY

E. J. MOURE,

BORDEAUX.

DISEASES OF THE TONSILS.

ACUTE TONSILLAR AFFECTIONS.

Before entering upon the study of the inflammations of these organs we must recall the fact that in addition to the two symmetrically placed glands to which the name tonsils has been given, there is another important collection of closed follicles occupying the base of the tongue, between the V-shaped line of papillæ and the anterior surface of the epiglottis. This collection of follicles has received the name of lingual tonsil or fourth tonsil. This impotrant fact being noted, we shall divide inflammations of the tonsil into superficial anginas, comprising catarrhal (follicular), ulcerative, herpetic, and pseudomembranous, and deep or parenchymatous phlegmonous anginas.

Acute Catarrhal Tonsillitis.

Synonyms.—Acute follicular tonsillitis, acute catarrhal angina.

Etiology.

Among the predisposing causes of acute inflammation of these parts we must first of all mention the age of the subjects; the affection is most common in adolescents and adults, but is rare in the aged. This fact receives its very natural explanation in the notable diminution of lymphoid tissue in advanced life. One attack predisposes to subsequent ones.

In most cases simple acute angina is due to cold acting upon the throat either directly or indirectly, as from cold feet, etc. Certain traumatisms of the nasal mucous membrane (Ruault, Moure) may excite an inflammation of the throat, especially of the tonsils. The extension of an acute coryza to the pharynx is a fact of daily observation. Certain anatomical conditions, such as interference with nasal respiration or hypertrophy of the tonsils, are also causes which predispose to acute inflammation in this region.

There is no doubt that the slightest interference with the important function of the lymphoid tissue existing in the pharynx at the entrance to the digestive tract, whether by a traumatism, cold,

or any other agent, will suffice to create the opportune moment for the appearance of an inflammation, the characters of which will vary according to the nature of the microbic agent which happens to find here a soil prepared for its development. According as these pathogenic agents are arrested at the surface or penetrate into the substance of the tissue, will the angina be superficial or parenchymatous. Finally, we must not forget that a generalized infection may determine the appearance of a secondary angina, whatever may have been the port of entry of the infection (Bouchard).

Symptoms.

Acute simple tonsillitis usually manifests itself at the onset by a series of general symptoms such as a feeling of depression, stiffness, headache, chills of variable intensity, and fever which is often rather high at first but is usually of short duration—seldom longer than eight or ten hours. Very soon after the appearance of these symptoms the patient feels a certain discomfort in the throat, swallowing becomes difficult or even painful, the sensation being confined to one side or felt on both, according as the inflammation is unilateral or involves both tonsils. The affected part is seen to be reddened and a little swollen, and from the orifices of the tonsillar crypts is welling up a clear whitish exudation giving somewhat the appearance of herpetic vesicles. The velum of the palate and the uvula participate in some cases in the local inflammation.

The fever now becomes more moderate, and in children it usually subsides altogether by the end of twenty-four hours; but the pain increases, the movements of the head and of the neck are painful, and often indeed there is an actual torticollis. The voice is thick and nasal, the tongue is coated, the appetite is lost. At this period, that is, about the second day, the orifices of the crypts are covered with a whitish pultaceous exudate, the tonsil is swollen and prominent, its pillars are red and infiltrated as is also the neighboring surface of the velum. The deposits on the tonsils are removed with some difficulty, but they do not ordinarily form a continuous layer. When the inflammation has attacked the Eustachian tubes, we note pain in the ears, deafness, and tinnitus aurium of the affected side.

During the course of the fourth or fifth day of the attack the functional symptoms begin to subside, swallowing becomes less difficult, but the local improvement does not seem to be in relation to the relief felt by the patient. Very soon, however, the local symptoms diminish, the tonsil returns to its normal size, the tongue cleans, and the appetite comes back, unless, as happens not infrequently, the inflammation attacks the opposite side. In such a case, when all

trouble seems to be over, the symptoms detailed above all return, though usually in diminished intensity, and the disease is thus prolonged for two or three days more.

In children the onset is often more sudden than in the adult; there is often a violent fever accompanied by delirium, and Renault has reported a case in a child of five years in which the temperature rose to 42° C. (106.5° F.).

Recovery is the rule, but recurrences are frequent, sometimes even periodical; each new attack leaves the child with larger tonsils, and the gland often remains chronically inflamed with dilated crypts.

Complications.

Among the most frequent complications are suppurating adenitis and catarrhal or purulent otitis media. Mild attacks have been observed of infectious arthritis (Sallard), nephritis (Bouchard, Landouzy, and others), pericarditis, peritonitis, pneumonia, etc. Cases have been reported of paralysis of the palate and even paralysis of the extremities following simple diffuse angina; but some authors believe that such complications point to the presence of a true diphtheritic tonsillitis, without false membrane.

Prognosis.

This is ordinarily favorable except in the fortunately very rare instances of some of the complications just mentioned.

Diagnosis.

This is usually easy, and need not detain us; in the section on herpes of the tonsil, we shall refer to the diagnostic points between that affection and the one now under consideration.

Treatment.

At the onset of the disease it will be well to administer quinine in dose varying according to the age of the patient and the country in which he lives. Locally applications of aromatic vapors for a period of five or ten minutes morning and evening or three times a day, and alkaline gargles constitute the main treatment. I generally prescribe the following solution to be employed as a throat wash four or five times a day:

Borax,	
Bromide of potassium,	
Antipyrin, ...	āā 4 gm.
Pure glycerin, ...	50 gm.
Decoction of coca leaves, ...	450 gm.

This is to be diluted with a half a litre of warm marshmallow water. Hot poultices are to be applied under the chin. A light diet of eggs, milk products, etc., is advisable for the first two or three days. If we wish to act directly upon the inflamed gland or glands we may make applications from the onset of the disease of a solution of chloride of zinc, 1:30 or 1:20; this is an excellent antiphlogistic in the early stages. A little later we may obtain good results with applications of lemon juice, which can easily be made even in young children.

It will be well to warn the parents against the bad habit of wrapping up too warmly the throat of a child who has had one or more attacks of sore throat, for this custom results simply in keeping the surface of the neck constantly moist and renders the child more liable to catch cold. It is much better to toughen these children by a well-considered hygienic and tonic regimen.

Acute Inflammation of the Lingual Tonsil.

Instead of attacking the tonsils, properly speaking, the acute inflammation may invade the collection of closed follicles at the base of the tongue, constituting what is called preglottic tonsillitis. This affection, which is not uncommon, is often unrecognized by reason of an insufficient examination.

It is usually manifested by few general symptoms that would suggest the true nature of the affection; the local symptoms on the other hand often attain a considerable degree of severity; there is pain on pressure on the tongue and around the neck, and deglutition is also painful, but the voice is usually normal or occasionally a little stuffy. Examination alone will suffice to establish the diagnosis and enable us to see not only the inflammatory swelling but also the pultaceous coating, generally discrete, covering the lingual tonsil. The recurrence of these acute inflammatory attacks, going on in some cases to the formation of a limited abscess in this region, often ends by causing hypertrophy of the follicles which may occasionally reach an enormous volume.

The *treatment*, except when an abscess is formed, is the same as that of acute follicular tonsillitis. If there is a collection of pus it will be necessary to open it by means of a properly curved bistoury, or preferably of the galvanic knife, which leaves the incision patent and allows of a free discharge of the pus.

The present writer once saw a chronic abscess formed just anterior to the epiglottis, which was extremely rebellious to treatment, undergoing acute exacerbations at intervals.

Herpetic Tonsillitis.

Definition.—By this term is understood an acute inflammation of the pharynx, accompanied by an apparently grave general disturbance, and characterized by the presence of herpetic vesicles upon the tonsils, the velum palati, and the wall of the pharynx.

Etiology, Pathogenesis.

Predisposing causes are found in the presence of a soil upon which the disease grows, and in this respect arthritism, nervous hereditary and general weakness hold the first place. The malarial subject is, according to some authors (Chapman), more exposed than others to contract affections of an herpetic nature. It is impossible, however, to discuss satisfactorily the special etiology of herpetic angina; I regard it as one of the manifestations of herpetic fever, for I believe that this fever may have exanthematous as well as enanthematous localizations, differing as regards seat but identical in their nature.

The exciting causes are very few, but two being admitted at the present day, namely, cold, the action of which none disputes, and the ingestion of food which has undergone a certain degree of decomposition, a cause which is not so universally admitted.

We do not believe that herpetic angina depends, in all cases, solely upon a disturbance of innervation, as has been held by Barensprung and Gottfried Scheff, for, as we shall see presently, we find in the progress of the disease all the evidences of a microbic affection, even the contagiousness; it is quite common to find small epidemics of herpetic angina in a single house or in barracks. Up to the present time the bacteriological examination of the contents of the vesicles has given contradictory results; there have been found in the fluid sometimes pneumococci, sometimes the staphylococcus albus or aureus, the streptococcus, or other micro-organisms existing normally in the mouth. In the little false membrane which represents the empty cover of the vesicle Löffler's bacillus has been found rather frequently of late, and from this it has been concluded, very erroneously I believe, that an herpetic angina is simply a diphtheritic angina. The affection so far from conferring an immunity, seems to predispose to new attacks. It occurs with greatest frequency in adolescence and adult life.

Symptoms.

The onset is ordinarily as rapid as that of pneumonia. There is a fever of 39.5° to 40.5° C. (103° to 105° F.), headache is intense, and

there are anorexia, nausea, a thickly coated tongue, chills, an aching in the bones, and somnolence (Lasègue).

During the first day the throat affection is hardly perceptible, but by the second day it presents all the characters of extreme acuteness. The tonsils are red and swollen, the pillars of the fauces and the pharyngeal wall are swollen and also red, with certain points of a still deeper hue. Soon herpetic vesicles appear; they are much more frequent on the tonsils but may nevertheless be seen scattered in little groups on the neighboring parts, such as the pillars of the fauces, the uvula, and the posterior wall of the pharynx. The eruption is usually bilateral and often symmetrical, and in the great majority of cases discrete; sometimes it consists in a large number of vesicles lying side by side on the tonsil and giving to the gland a somewhat bristly appearance. The vesicle itself, the size of a millet seed, is first filled with a transparent liquid which escapes at the end of twelve to thirty-six hours through rupture of the envelope. It is surrounded by a small inflammatory area scarcely visible on the tonsils, the mucous membrane of which is normally quite red. After the evacuation of the liquid there exists a little ulcer, covered with a whitish coating or a pellicle of the same color, a veritable adherent false membrane which, coalescing with the pellicles of adjacent vesicles, may be mistaken for a true diphtheritic pseudo-membrane. Within four or five days the whole has disappeared leaving no traces. Sometimes the eruption occurs in successive crops with or without a new access of fever, and it is very rare that vesicles do not appear in other parts of the body, such as the lips, the openings of the anterior nasal fossæ, the internal surface of the cheeks, and even in the larynx (Brindel).

There is no glandular enlargement at the angle of the jaws, or at least if there is it is exceedingly slight.

Course, Duration, Termination.

The evolution of the disease is rapid and in from three or six days the whole has terminated. The defervescence is rapid and the patient finds himself cured almost without any period of convalescence; this, at least, is one of the most usual modes of termination, but if the eruption occurs in several crops the disease may last longer, reaching even fifteen to twenty days. The attacks of herpetic angina which some individuals suffer from after simple digestive disturbance, or which certain women have at the time of menstrual periods, are less grave, of shorter duration, and less painful, but may recur at intervals for months or years.

Prognosis.

This is good. There is really but one possible complication, that is the engrafting of a diphtheritic angina upon an herpetic sore throat at some period of its evolution.

Diagnosis.

At the beginning an herpetic angina may be confounded with simple acute tonsillitis, erysipelas of the pharynx, a rheumatic or a scarlatinal angina. This confusion, however, cannot last more than one day, for then the appearance of the vesicles will remove all doubts. The only diagnosis of importance or of special difficulty is that between the disease under consideration and diphtheritic tonsillitis. Bacteriologists say that this is very simple and that all that is necessary is to cultivate a small piece of the doubtful false membrane in order to obtain indubitable results by the end of twenty-four hours. Without wishing to deny that a certain number of cases of diphtheritic angina may have been taken for herpetic tonsillitis, I believe that it is, up to the present at least, impossible for the clinician to accept all the deductions of the bacteriologist, for it has been shown that the Klebs-Löffler bacillus exists in many parts of the buccal cavity in healthy individuals. It is necessary, therefore, to take into account the clinical facts and to be able to associate them with the results of the bacterioscopic examination.

The presence of small discrete white points, two to four in number on each tonsil, the coexistence of labial herpes, intense pain on swallowing, an acute onset with fever, dating back not more than from twenty-four to forty-eight hours, and finally, the absence of cervical adenopathy will point to the presence of herpetic angina and will enable us to establish the diagnosis with considerable certainty, at any rate that of the primary lesion. We know, however, that a diphtheritic sore throat may become engrafted upon herpes of the pharynx; in such a case we see the false membrane extend, fall off, and then be reproduced, adenopathy develop, the patient become pale, and the urine contain a certain amount of albumin.

In all cases of doubt, however, we ought to act as if we were dealing with a case of diphtheria, especially if a bacterioscopic examination has been made and the Klebs-Löffler bacillus found, either alone or associated with some other micro-organism.

Treatment.

An emetic and a cathartic administered at the onset of the disease will often relieve the headache and diminish considerably the dys-

phagia. Sulphate of quinine and antipyrin will reduce the fever and certainly shorten the duration of the disease.

Locally gargles of borax and bromide of potassium and washes of boric acid or weak carbolic water (three to five per cent.), or painting with carbolized glycerin (two or three per cent.) have the advantage of reducing the tonsillar inflammation and preventing the engrafting of a diphtheritic sore throat upon the soil already prepared for it.

Acute Ulcerative Lacunar Tonsillitis.

I shall describe under this name a special form of acute inflammation of the tonsil characterized by the presence of large ulcers resembling closely tertiary syphilitic ulcerations, with which they are ordinarily confounded.

Symptoms.

Lesions of this kind are usually not accompanied by any well-marked febrile reaction; most frequently indeed the beginning of the trouble is insidious, passing almost unperceived, and when the patient first notices the difficulty or pain on swallowing, the lesion will be found to be already present. For this reason the occasion has very rarely presented itself of studying the first appearances of the ulceration and of determining whether the supposed pathogenesis is absolutely exact.

When the patient seeks advice we find usually on one of the tonsils, sometimes on both at the same time, a grayish ulceration, covered with a cheesy exudate which can be readily removed by a brush or pledget of cotton, leaving a sanious, mammillated reddish surface composed of little granulations, sometimes even actual polypoid excrescences, as shown in the accompanying illustrations (Figs. 33 to 36). The borders of the ulcer are clean-cut, red, inflamed, but not particularly swollen, and the rest of the tonsil does not appear to be inflamed, being only slightly increased in volume. We have here to do with a purely local lesion affecting only one part of the tonsil, seeming to take its origin in one or several of the crypts, without encroaching upon the neighboring parts. At first sight one is reminded of a cauterization made with the thermo-cautery in a very limited space, which has progressed to the point where the scar is about to fall off—that is to say, three or four days after the cauterization has been made. But it much more resembles in its general appearance the lesion left after localized ignipuncture.

Very often the tongue is a little coated and the breath is tainted without being absolutely fetid. It is not rare to find two or even

three ulcers upon the same tonsil, separated by areas of glandular tissue, which are scarcely at all inflamed. These ulcers develop each by itself, without showing any tendency to coalesce. Often they may be seen to be in different stages of development, or one will be nearly healed while the other is in full course of evolution. We

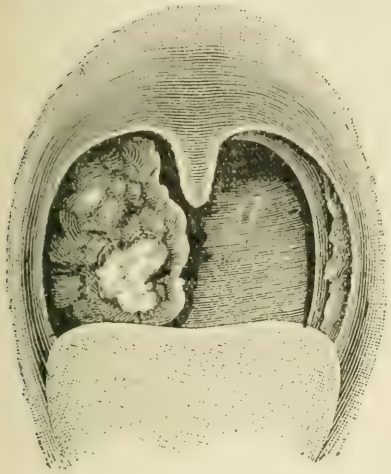


FIG. 33.

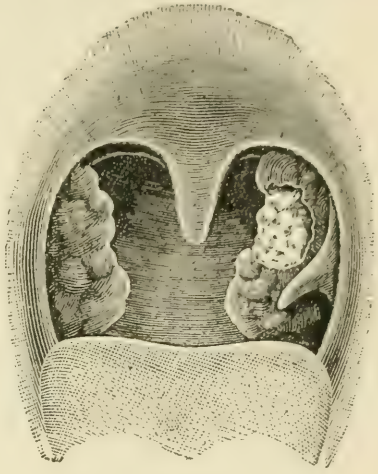


FIG. 34.

FIG. 35.

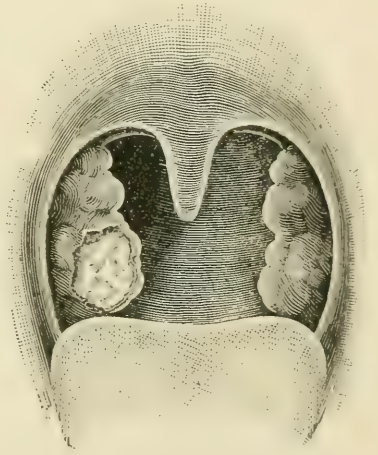


FIG. 36,

FIGS. 33-36.—Acute Ulcerative Lacunar Tonsillitis.

have not to do in these cases with the little cup-shaped, almost microscopic ulcers which have been described under the name of acute lacunar tonsillitis, but rather with extensive losses of tissue which may attain the size of a ten-cent piece or even larger. Their shape appears to me to be very variable, but generally, although the

edges are ragged and irregular, the lesion as a whole is more or less oval or round, as if it had been made by a gouge acting upon a rather soft tissue which is torn rather than divided. The ulcer is always rather deep and in some cases we have seen it extending down to the floor of the tonsillar space. The pillars have almost always escaped in the cases which we have observed. They may, however, be very slightly reddened and infiltrated, if the ulcerated gland is rather large and adherent to the pillar. Although the lesion is a local one the patients have usually more or less lassitude and are a little anæmic; but we seldom observe any fever or other severe general symptoms.

There is usually no swelling of the submaxillary glands; and in the exceptional instances in which glandular enlargement exists the affected glands are few in number, not more than one or two, and painful to the touch. Often pressure in the neck at a point corresponding to the affected tonsil elicits a rather sharp pain. These functional troubles are generally of short duration and are observed only during the period of formation of the ulcer, disappearing gradually with the latter. The lesion is absolutely benign and ephemeral, running its course in a few days apparently spontaneously like an acute affection. Although recurrences are possible they do not appear to be the rule, for we have had occasion to note the return of the ulcer but once and that some months after the first attack and upon the opposite tonsil.

Diagnosis.

What we have just said of the symptoms of this form of ulceration would seem to dispense us from dwelling long upon its differential diagnosis; but the affection is so little known and has been so often mistaken for other forms of ulceration that it seems incumbent upon us to say a few words concerning the differential diagnosis between it and chancre or ulcerating gumma of the tonsil.

There is no resemblance whatever between these ulcerations and acute tuberculosis of the tonsil; I have never seen a case in my practice nor read of any in the literature on the subject, particularly in Barth's thesis, which resembled even distantly the affection which we are studying at present. Lupous ulceration (chronic tuberculosis) occasionally presents some points of resemblance; but, apart from the fact that lupus is not limited to a particular point of the tonsillar tissue, its borders are always more or less red and infiltrated, its contours are less distinct, and the surface of the ulcer is covered with pinkish granulations, gradually disappearing towards the edges. The progress of the disease is slow, insidious, and intermittent and its

duration is always rather long, often several months. Moreover, primary lupus confined to the tonsil is rather rare, if we may judge from the few cases reported in literature.

We may also eliminate the danger of confusion with cancer or other malignant tumor of the tonsil, the macroscopical characters of which and especially the clinical features possess nothing in common with those of this acute ulceration.

Pseudo-membranous or herpetic tonsillitis or other acute inflammatory angina is marked by very characteristic symptoms, which will prevent confusion with this purely local lesion.

Chancre of the tonsil to which Fournier and Dieulafoy have recently redirected attention, and of which numerous examples will be found quoted in the thesis of Pivaudran, bears but a faint resemblance to simple ulceration; the signs of local infection are here most distinct, the entire tonsil is red and swollen, and the velum palati and anterior pillar participate very often in the infectious process. The voice is thick, and the submaxillary glands are enlarged and present all the appearances characteristic of symptomatic adenitis. Moreover, chancre of the tonsil is of quite long duration, often several weeks. It is sufficient to have seen this lesion once in order never to be in danger of confounding it with any other affection of this region, without mentioning the later symptoms of constitutional involvement which will suffice to clear up any doubtful cases. I need only mention mucous patches, which even in smokers are always more diffuse, more superficial, more salient, and more obstinate than the lesion which we are now considering.

Ulcerated gummata are, perhaps, of all possible lesions of the tonsil those which may bear most resemblance to acute ulcerative lacunar tonsillitis; it was this lesion which I used always to think of formerly when I knew little or nothing at all of these transitory ulcerations. The gummy ulcer, indeed, presents the appearance of a clean-cut loss of substance with a yellowish, pultaceous coating which, after removal, leaves a sanious, reddish, fungating surface; but the characteristic feature of the gumma is that it does not come in the centre of the tonsil, but rather occurs usually toward its edges, either in front or behind, above or below, so that it invades very rapidly either the pillars, the velum, the pharynx, or the base of the tongue, extending both in area and depth. Tertiary syphilis of the pharynx, when untreated, spreads in every direction and gives rise to extensive destruction of tissue, which leaves behind it the well-known cicatricial adhesions. The edges of the syphilitic ulcer are always more or less red, inflamed, and infiltrated, features that are never present in simple ulceration. To these very marked character-

istics we may add the slower course and consequently longer duration of gumma, to say nothing of its irregular form and the antecedents of the patient which may be of service in establishing the diagnosis. The age of the patient is also of importance in a diagnostic sense, for the gummatous ulcer is frequent above the age of thirty, while lacunar tonsillitis is more common in persons between the ages of eighteen and twenty-five. The latter has the frank onset of an *angina a frigore;* gumma commences more insidiously and with less rapidity.

Etiology, Pathogenesis.

Acute ulcerative lacunar tonsillitis appears to be more frequent in the spring and autumn, and, judging from the cases that have come under my own observation, affects chiefly persons of from twenty to thirty years of age. I have never seen it in children nor in adults over forty years of age. Its relative infrequency after this age finds a ready explanation in the fibrous transformation which the tonsillar tissue undergoes at this period which renders it less prone to acute inflammations.

The lesion occurs usually in those who are debilitated or fatigued, in whom it may appear in consequence of the slightest chill. I have very often seen this local affection accompany the evolution of a wisdom tooth, which was being cut with difficulty; at other times I have noticed it occurring after excessive fatigue or after an attack of influenza.

Some of the patients, but not all, had tonsils of the type described as lacunar; the lacunæ were often numerous and deep on the opposite tonsil, and frequently also the glands were of such a size as to deserve the qualification "hypertrophied." On the other hand I have seen these lesions on tonsils which were not at all large, scarcely projecting beyond the anterior pillars.

There can hardly be any doubt that the ulcerative process is of microbic origin, although we have never been able in the pathological laboratory of Bordeaux to discover any micro-organisms in numerous sections colored by Gram's and Weigert's methods. According to Legendre and A. P. Claisse, "the various forms of tonsillitis, always an infectious disease, may be due to the action of microbes of the most varied sort, and from the external appearance, except perhaps in case of diphtheria, we can hardly tell what is the pathogenic agent, whether streptococcus, staphylococcus, or pneumococcus."

Nevertheless, while admitting the microbic nature of tonsillar ulceration, we believe that the cause must be looked for in acute inflammation of the crypts, the desquamative products of which, too

rapidly exuded, may not be promptly enough eliminated. The result of this is an excessive dilatation of one or several of the lacunæ, and their walls being broken down, they form a single cavity filled with epidermic detritus, which gradually breaks down the outer wall, just as would pus; when this external layer is once destroyed we find an ulcer with ragged edges without any signs of true diffuse inflammation and without any other marked symptoms. Histological examination would seem to confirm this view.

Treatment.

The surface and the cavity of the ulcer should be thoroughly cleansed from all the sloughing products there present. This is accomplished by means of a washing or rather sponging out, made with a pledget of cotton moistened with a solution of chloride of zinc, 1 : 20 or 1 : 30. The following is a useful formula for this solution:

Hydrochlorate of cocaine,	0.3 to 0.5
Chloride of zinc,	1.0
Pure glycerin,	10.0
Distilled water,	15.0 to 20.0
Hydrochloric acid,	1 to 2 drops.

When this is done the patient may gargle several times a day with a borax or bromide of potassium solution in glycerin and water. The following day the ulcer may be again cleansed with the chloride of zinc solution or with the iodine and carbolic-acid solution. Plain carbolized glycerin, 1 : 10 or 1 : 15, constitutes an excellent application.

When the ulcer has begun to cicatrize we may omit these applications and prescribe simple gargles of alcohol and water. Scraping the ulcer with a curette has often given me very good results, when done early.

The most important part of the treatment consists in local hygiene in order to prevent contamination of the neighboring parts, as apparently happened in the case of one of my patients. A cure within a period of a few days is the rule in these cases. It is needless to add that if the patient is a smoker he ought to abstain until the ulcer is cured.

Non-Diphtheritic Pseudo-Membranous Angina.

Definition.—Under this term are included inflammations of the throat marked by the production of false membranes not due to the presence of the Klebs-Löffler bacillus. At the present day, when the bacterioscopical examination of anginous products has become a matter of almost daily routine, we very often see pseudo-membranous

sore throats resembling macroscopically true diphtheria which can be differentiated from it only by bacteriological methods.

Etiology and Pathogenesis.

Pseudo-membranous angina, which is fairly common, is due most frequently to the presence of the streptococcus, of the pneumococcus, or even of the bacterium coli commune, so that there is no special pathogenic micro-organism of the disease. The etiology is much the same as that of other inflammations of the throat, the chief exciting causes being atmospheric changes, damp cold, surgical operations upon the tonsils, and lesions of neighboring regions, the patient being already predisposed by reason of a lowered general condition. Some of these anginas are distinctly contagious.

Symptoms.

Diphtheroid anginas are primary or secondary. The former resemble closely true diphtheria; their onset may be sudden and marked by a chill, and the fever, as in diphtheria, may be slow and insidious. The false membrane appears first on one tonsil, covering it entirely, and thence spreading to the pillars, to the pharynx, and to the opposite tonsil, enveloping the uvula as in a sheath, is reproduced after removal, and is found to cover an erosion, sometimes even an ulcer of the mucous membrane. Adenitis is more or less marked, the breath is fetid, but the general condition remains ordinarily good.

The evolution of the false membrane takes four or five days, after which time it is not reproduced, the fever falls, and the tonsil becomes nearly normal in appearance, though still a little redder than ordinary. Relapses are not uncommon.

Primary diphtheroid tonsillitis may, however, assume a much more alarming form, resembling in all respects malignant diphtheria, with extreme fetor of the breath, a muco-purulent discharge, adenophlegmon of the neck, prostration, elevated temperature in the adult, the fatal termination coming on in two or three days. This form is said by Epstein, of Prague, to be not very infrequent in marantic infants. In the more fortunate of these cases recovery takes place after a rather long convalescence. The grave forms of angina are due to the action of staphylococci or of streptococci.

The secondary diphtheroid anginas are met with in variola (rarely), in measles, and most frequently in scarlatina. There are two periods of the disease in which pneudo-membranous angina may be encountered: at the very beginning and during convalescence. The late anginas are almost always diphtheritic, the others rarely so; in the

latter case the mucous membrane around the false membrane is red and beneath the latter it is sanious and ulcerated. The general condition of the patient may be very grave and the angina itself may be benign, severe, or extremely malignant (hypertoxic angina).

The onset of diphtheroid chancre may be announced by very disturbing general symptoms (Fournier), and the differential diagnosis between it and diphtheria may present serious difficulties; indeed it may be only by watching the progress of the affection, or upon the appearance of some other symptom, such as roseola, that we can in the absence of a bacterioscopic examination distinguish between the two.

Complications.

The invasion of the larynx by non-diphtheritic pseudo-membranes is not very frequent, but on the other hand the naso-pharynx, the nasal fossæ, and even the face (erysipelas) are more often attacked than in true diphtheria. The pulmonary complications, such as bronchitis and bronchopneumonia, are hardly ever seen except in the malignant cases. Œdema of the glottis is sometimes seen, but much more frequent complications are extension of the inflammation through the Eustachian tube to the tympanum and adenophlegmons of the neck (latero- and retropharyngeal abscesses) with all their consequences. The infectious matter may be transported to a distance, giving rise to acute nephritis, arthralgia, suppurative arthritis, erythema, purpura, and paralyses identical in appearance with diphtheritic paralysis. In exceptional cases we have inflammation of the larger serous membranes, the pleura, pericardium, peritoneum, and meninges.

Diagnosis.

This is often established by the bacterioscopic examination; yet the diagnosis is sometimes difficult. Every false membrane of the tonsils and pharynx which does not contain the Klebs-Löffler bacillus is certainly not diphtheritic, but the converse is not true, as we have shown when speaking of herpetic tonsillitis.

Treatment.

This is to be carried out on the same lines as that of herpetic angina, but we may endeavor also to obtain intestinal antisepsis by means of naphthol, benzo-naphthol, or salol. The patients should be isolated as strictly as possible until the mucous membranes have returned to their normal appearance. A relapse may often be prevented by tonsillotomy if there is hypertrophy of the glands.

Diphtheritic Angina.

Definition.—By this term is understood an inflammation of the throat characterized by the presence of false membranes which are themselves due to the presence of the Klebs-Löffler bacillus—in other words, a localization of diphtheria upon the mucous membrane of the tonsils and pharynx.

History.

Bretonneau (1818–1826) distinguished diphtheritic from gangrenous angina, and recognized the identity of the different localizations of diphtheria. Trousseau taught that it is a general disease with special localizations. The Germans distinguished what was for them a gangrenous and infectious angina from croup, which they regarded as a purely inflammatory exudate, until Klebs (1883) and Löffler (1884) brought forward by their discovery of the specific microbe the absolute demonstration of the unity of the disease. Roux and Yersin (1888–1890) isolated the toxin of the Klebs-Löffler bacillus and by injecting it into animals succeeded in obtaining lesions identical to those resulting from injections of pure cultures of the bacillus itself.

Etiology and Pathogenesis.

Primarily diphtheritic angina is a strictly local affection; the bacillus remains in the neighborhood of the points where the false membrane is developed, but it produces in this situation a toxin which is absorbed by the organism and becomes the occasion of more or less dangerous symptoms. Moreover, in addition to the Klebs-Löffler bacillus we often find other microbes, the action of which is added to that of the first and modifies the course and prognosis of the disease.

One word as to the bacillus. It is met with on the surface of the false membrane and is a straight or curved rod, immobile, twice as thick as the tubercle bacillus and of about the same length—that is the appearance which it presents when cultivated on gelatinized serum, but on false membrane it has rather the shape of a pear or a club. It stains readily. The following is the method employed in the examination for this bacillus. A small fragment of the false membrane is removed by means of a sterilized platinum loop, or some of the material scraped from the surface may suffice. Two or three gelatinized serum tubes are immediately sowed with the matter so collected and are then placed in an oven at 37° C. for seventeen or eighteen hours. At the end of this time little round colonies of a

grayish-white color with an opaque centre may be seen on the surface of the serum; one of these colonies transferred to a slide and colored by Löffler's blue will show the presence of bacilli having the appearance described above. It has been shown that the longer the bacillus is the more virulent it is, so that in the report of the results of this examination one ought to note whether the bacillus is short, medium, or long. Another method for determining the virulence of the microbe is to obtain pure bouillon cultures and inoculate guinea-pigs with a few drops; these animals are very sensitive to the action of the bacillus and will die at the end of thirty to forty hours if the microbe is that of diphtheria.

The microbes most frequently associated with the Klebs-Löffler bacillus are the staphylococcus cyaneus and aureus and especially the streptococcus pyogenes the presence of which almost always gives to the affection an exceptional gravity and very rapid progress; it is to the streptococcus especially that we ascribe the secondary affections, such as phlegmons of the neck, suppurative adenitis, arthritis, bronchopneumonia, etc. The pneumococcus of Talamon-Fraenkel is also encountered, but less frequently than the micro-organisms above mentioned.

We have, therefore, a pure diphtheria and a polymicrobic diphtheria, although in practice it is often difficult to differentiate the two; but the serum therapy of diphtheria has now invested this distinction with a new importance.

The chemical characters of the toxin are not yet clearly established; according to some (Brieger and Fraenkel) it is a toxalbumin, for others (Gamaleïa) a nucleoalbumin, and for others yet (Roux and Yersin) a substance allied to the diastases.

Diphtheritic angina is an epidemic infectious disease, not conferring immunity, and occurring most frequently in young children. Its contagiousness may precede the appearance of the false membranes; it is at its maximum during the entire period of the false membranes, but it may persist for one or two months after their disappearance. The latter assertion is in accord with the results of bacterioscopic examination which demonstrates during this entire period the presence of virulent bacilli in the throat. Contagion occurs through the air, clothing, the hands, bedclothes, food, the false membranes, etc. The question of soil is an important one in the development of the disease, and all acute or chronic inflammations of the pharynx, of the tonsil, of the nose, and of the naso-pharynx, the presence of enlarged tonsils or of adenoid vegetations all favor the growth of the bacilli. When we add to this a poor general condition and defective hygienic surroundings we shall have the best conditions for the development

of cases which are isolated at first but which soon increase in number to the proportions of an epidemic.

Pathological Anatomy.

The false membrane in diphtheria differs from non-specific pseudo-membranes only in the presence of the Klebs-Löffler bacillus. It covers the mucous membrane, replacing the superficial layers of the epithelium and sometimes the entire thickness of the latter. It is composed of a fibrinous reticulum in the meshes of which are found some lymphatic cells, red blood corpuscles in small number, and degenerated epithelial cells the nuclei of which will no longer stain with picrocarmine. The corium of the mucous membrane presents all the characters of an acute inflammation; it is infiltrated with a large number of round cells and its vessels are dilated. The older the false membrane is the thicker it is; it is more or less white in color, and the mucous membrane beneath it bleeds easily; it is sometimes removed with difficulty.

The entire lymphatic system may show signs of acute inflammation (swelling) corresponding to a considerable infiltration of leucocytes in the follicles and an enormous dilatation of the blood-vessels; the submaxillary and carotid glands are ordinarily the first affected. The inflammation frequently extends outside of the gland itself into the neighboring cellular tissue. Peyer's patches undergo the same alterations as the rest of the lymphoid tissue. The blood is dark in color, coagulates feebly after death, and contains a large proportion of white globules. The spleen is enlarged, black, and infiltrated with a large number of round cells. The liver is congested and infiltrated with round cells, and these endothelial cells as well as the hepatic cells themselves are filled with fat (Morel).

The changes in the kidneys vary in degree but are constant; they consist in an acute inflammation, characterized by tumefaction of the endothelial cells of the capsule, exudation between the capsule and the glomeruli, infiltration of white cells along the course of the straight tubes and of the vessels, and desquamation of epithelium into the convoluted tubules. The heart is dilated and there is often acute myocarditis, degeneration of the muscular fibre, infiltration of round cells in the connective-tissue cracks, and endoperiarteritis of the arterioles.

The central nervous system may be affected—acute myelitis of the anterior cornua and inflammatory changes in the anterior roots (Roger and Damaschino); but the peripheral lesions are the most marked—the peripheral neuritis described by Charcot and Vulpian (1862), by Déjerine, and by Gombault (1880). The paralyzed muscles undergo

granulo-fatty or waxy degeneration. The atrophy which results from this degeneration may be permanent, as has been observed especially in the larynx by Rokitansky, Charcot and Vulpian, and others.

At the autopsy of those dead of diphtheria we meet lesions which cannot be attributed to the presence of the Klebs-Löffler bacillus, but rather to secondary infections; these are gangrene, suppurative tonsillitis, adenophlegmons, bronchopneumonia, pericarditis, in rare cases meningitis, suppurative otitis media and interna (Moos), suppurative arthritis, and generalized infections.

Symptoms.

The study of the clinical forms of diphtheria, which was in a very confused state, even before the latest bacteriological discoveries, has become still more so since the examination of all pseudo-membranous anginas has been systematically made. If indeed we characterize as diphtheritic all anginas in which the Klebs-Löffler bacillus is found we shall see that a number of benign pharyngeal lesions, such as certain herpetic anginas, must be included under the term "diphtheria" and go to increase the number of clinical varieties of the disease. We shall adhere to the old clinical division of a benign form, to-day called the monomicrobic; an infectious form, the bacillococcic; and a toxic, or bacillostreptococcic.

1. Benign or Monomicrobic Angina.—The onset is ordinarily insidious; the period of incubation varies from one to eight days. The local symptoms are not always the first that are noticed; there may be a little headache, general malaise, a slight elevation of temperature, loss of appetite, one or two attacks of vomiting, a coated tongue, a little diarrhœa, and eyes abnormally bright; this is the usual mode of onset of the disease. The patient does not complain of his throat until the next day or the day after, and even then the pain of which he complains is very slight; the cervical glands begin to enlarge, however, and increase rapidly in size, but remain perfectly distinct one from the other. During the succeeding days the patient becomes pale and depressed, the conjunctivæ are white, the appetite is lost entirely, but the fever remains moderate; the pain in the throat may not increase, but we frequently find that the voice becomes nasal two or three days after the beginning of the affection, drawing attention to the throat, which, moreover, we should never neglect to inspect, especially in children, upon the appearance of the slightest morbid symptom.

Although this is a frequent mode of onset of simple diphtheria, we may find, even in the same epidemic, varieties which differ

absolutely in appearance although bacteriologically they are identical. For example, we sometimes see in the first days of the disease an intense fever, convulsions (in children), very severe sore throat, chills, etc., all of which symptoms decline or even disappear at the end of two or three days, the further course of the disease assuming a benign form.

If we examine the throat we may, even on the first day, see a little redness on one or the other tonsil, but nothing more. At the end of twenty-four to thirty-six hours, however, one or two whitish points are formed which rapidly coalesce, extend in area, and form a white pellicle which is thinner on the periphery than in the centre and rather adherent; this is the pseudo-membrane. If we remove it carefully the subjacent mucous membrane is not found to be ulcerated, but the false membrane is very rapidly reproduced, often in the course of two or three hours. From the tonsil, where it first appears, it rapidly passes to the pillars of the fauces, to the uvula, which it may envelop like the finger of a glove, to the pharynx, even to the velum palati and the tongue. If no abnormal complications ensue at the end of from five to eight days the false membrane, even when not treated, is no longer reproduced, and the patient enters upon convalescence, if he has escaped the very serious dangers of poisoning by the microbic products and of asphyxia through the mechanical impediment to respiration. The first depends upon the number of bacilli present in the pharynx and also upon the resistance which the organism opposes to their development and to their products of secretion. Albuminuria, which is less frequent in this form of diphtheritic angina than in the other varieties, indicates almost always that a large amount of the microbic products has been absorbed. The second danger will be referred to at the end of this section, when we come to speak of the complications of diphtheritic angina.

2. *Infectious, Polymicrobic, or Bacillococcic Angina.*—The onset of this form often resembles that of the one just described, but the local and general symptoms quickly become more marked; the facies changes rapidly, although the intelligence remains intact, as in the preceding case; the face is pale, the ganglionic swelling of the neck is more pronounced, and albuminuria is more frequent. The false membranes grow very rapidly in all directions and have a greater tendency than in the preceding form of the disease to invade the neighboring cavities, nasal, buccal, and laryngeal. The mucous membrane in the neighborhood of the false membrane is more congested, there is a muco-purulent discharge from the nose, and a bad breath; the fever increases; the patient is more profoundly poisoned than in the simple form, and the complications in the course of the disease

and during convalescence are more frequent and more grave. The progress of the affection is slow, and in favorable cases the disease often takes fifteen to thirty days or even several months to run its course. It is here especially that we find the greatly prolonged forms of diphtheria.

3. *Toxic or Bacillostreptococcic Angina.*—Not all cases of sore throat in which we find the streptococcus in addition to the Klebs-Löffler bacillus are toxic, and this is fortunate for, according to recent investigations, the association of these micro-organisms is much more common than was formerly supposed; yet the more frequent recognition of this association does not correspond to any increase in the gravity of diphtheritic sore throat in general; all that we can say with certainty is that in the very severe toxic forms of diphtheria we almost always meet with the streptococcus pyogenes in abundance. This need not surprise us when we remember that in a tolerably large number of malignant affections, such as erysipelas, puerperal fever, many cases of abscess, and some bronchopneumonias, this is the only or chief pathogenic agent.

From the very onset the malignancy may be marked by profound depression, a typhoid condition, an excessive ganglionic swelling with implication of the surrounding cellular tissue, a small and rapid pulse, a high temperature, and a scanty secretion of urine containing a large proportion of albumin; the patient dies in from twenty-four to forty-eight hours. In other cases the advance of the disease is less rapid and may consume five or six days; after an onset in one form or another the disease may suddenly assume the characters of great malignancy, the general condition becomes rapidly bad, the pulse is thready, the temperature falls below the normal; the cervical glands are as large as in the preceding form, the false membranes exhale a repulsive odor, the mucous membrane yields to the slightest touch; spontaneous hemorrhages occur from other mucous membranes (of the mouth, stomach, and intestines) in the kidney, and in the bladder, and albuminuria is constant; the patient dies in collapse.

In other cases, again, the false membranes may be hardly perceptible while the adenitis remains marked, and the membrane may even disappear, notwithstanding the persistence of a bad general condition; the patient gradually falls into a collapse from which he does not emerge. According to Bourge there may even be insidious cases of toxic diphtheria analogous to those of walking typhoid, which present no symptoms other than cervical adenitis and pallor of the integuments, and which terminate rapidly in death, preceded or not by collapse.

Complications.

An exaggeration of the local symptoms may produce gangrene of the pharynx (tonsils, velum palati, uvula, or posterior wall of the pharynx). The extension of the false membranes to the mucous membrane of the mouth constitutes buccal diphtheria; the pseudomembrane may be found on the inner surface of the lower lip, the labial commissure, palatine vault, or the tongue. Other localizations of diphtheria have been seen in the œsophagus, the stomach, perhaps the intestines, but freq ently the anus. The false membrane may invade the naso-pharynx and the nasal fossæ, extending from the first location to the middle ear, and from the second to the conjunctiva. It very commonly invades the larynx and causes croup; but we need not dwell upon this nor upon the bronchopneumonia, which is also a very common sequel.

The external genital organs may become the seat of diphtheria; this involvement is observed especially in the severe secondary forms.

The skin may be affected in one of two ways: false membranes may be formed upon the slightest abrasion (acne, chaps, eczema, psoriasis) and extend thence, especially in the severe forms, over the healthy skin; at other times it may become the seat of erythema (scarlatinal form, rubeolic, vesicular), œdema, and, even when no albuminuria is present, purpura and subcutaneous ecchymoses.

An exaggeration of the inflammation of the glands, especially in the polymicrobic and streptococcic forms, results in suppurative adenitis, developing as a circumscribed adenophlegmon, the evolution of which is but an epiphenomenon in the course of the diphtheritic angina. During convalescence we sometimes see secondary infectious arthritis, simple or suppurating.

The cardiac complications (acute myocarditis) are more grave; they come on during the course of the disease or in convalescence, often passing unperceived in the child, whose sudden death they may cause, being indicated in the adult by modifications of the pulse and of the apex beat and by syncope.

Albuminuria, which is transitory in the great majority of cases, is very frequent in polymicrobic diphtheria. The renal alteration of which it is the expression seldom results, however, in uræmic complications.

All the cases of sudden death observed during convalescence from diphtheria are not necessarily due to a lesion of the myocardium or of the kidneys, but are sometimes the result of nervous lesions, which, beginning as paralysis and anæsthesia of the velum palati, may involve successively the muscles of the pharynx, of the œsophagus,

the larynx, the tongue, the lips, the cheeks, the motor muscles of the eye and those of accommodation, the muscles of the extremities, the intercostals, the diaphragm, the sphincters, both vesical and rectal, and finally, fortunately in rare cases, the cardiac muscle. Whether early (from the fifth to the eighth day) or late (from the fifteenth to the thirtieth day), the paralysis is ordinarily bilateral and symmetrical, of short duration, and accompanied sometimes by anæsthesia. When generalized it may last from three to six or eight months.

Course, Duration, Termination.

A spontaneous cure of diphtheria is much more frequent than is generally supposed, if we add to the anginas formerly called diphtheritic all those in which the Klebs-Löffler bacillus is present. Death may occur in several ways: suddenly in consequence of myocarditis or of pneumogastric paralysis, more slowly through intoxication, through invasion of the respiratory passages by croup, through bronchopneumonia, secondary infection, or more rarely, as a result of the induced cachexia. Relapses are frequent, either a few days or at a considerable interval after the first attack.

Prognosis.

We cannot be too careful in our prognosis in any case of diphtheria, for if the patient escapes from the disease itself he may succumb to one of the complications. However, when we see false membranes with little tendency to spread and to return after removal, a slight adenitis, a good general condition, and no marked depression, we may hope for a benign course of the disease, if the patient is placed under good hygienic conditions. The prognosis of diphtheria, we may add, has been greatly improved since the introduction of serum therapy.

Diagnosis.

To dwell at length upon the points of diagnosis would be a pure waste of time, since the bacterioscopic examination alone can give a certain result. Furthermore, what we seek to know to-day is simply whether there is or is not present the bacillus of diphtheria and whether the disease is a proper one for the employment of serum therapy. We should, however, remember that there are cases of angina with very slight or even absent pseudo-membranes which are none the less diphtheritic.

Treatment.

The limits of this article compel us to be brief, so we shall speak simply of the measures employed at the present day and not of

those that were formerly in vogue. The patient ought to be at once isolated in a well-lighted room, in which there is a constant evaporation of antiseptic substances, such as creosote or a carbolic-acid solution, in order that the atmosphere may be at the same time moist and antiseptic. The patient must be nourished in such a way as to maintain his strength and permit him to contend successfully against the debilitating disease. The local treatment has become much less important since the introduction of serum therapy; and instead of removing the false membrane often by force, causing the mucous membrane to bleed, and then applying strong antiseptics, such as carbolized glycerin, perchloride of iron, tincture of iodine, salicylic acid, etc., many practitioners to-day content themselves with washing the throat by means of a large syringe with a mild solution of carbolic acid, salicylic acid, boric acid, or chloral or with lime water. They do not worry on account of the false membranes, which fall of themselves during the treatment by serum. We believe it wise, however, not to neglect altogether local applications in the case of polymicrobic diphtheria.

As soon as the diagnosis of diphtheria is established, or even earlier in a suspicious case, with grave symptoms, the present practice is to give a subcutaneous injection of diphtheria antitoxin. By means of a suitable syringe, that of Roux for example, we inject 10 c.c. under the skin in the abdominal or natal region. In children under one year of age we may inject 1 c.c. for each month of the child's age; in children over two years of age we may, without danger, inject 20 c.c., but ordinarily 10 c.c. will suffice; this dose may be repeated on the next day or the day following that, if the disease does not show signs of improvement. Under the influence of these injections the general condition improves, the temperature decreases, the false membranes fall off of themselves without being reproduced, and convalescence ensues quite rapidly.

We may say a word as to the occasional inconveniences of this method; we observe after an injection more frequently than before this treatment was introduced various forms of erythema and rarely of purpura, sometimes arthropathy, but more especially urinary and circulatory disorders (increased secretion of urea and of phosphates, diminution of the chlorides, rarely acute nephritis, tachycardia, or arhythmia), and paralysis. Of all these accidents erythema is the most frequent, but it is of slight importance and need not deter the practitioner from the employment of preventive inoculations during the course of an epidemic.

This method of treatment is still being developed and we need not dwell longer upon it in this article. We may, however, add a word

concerning the results thus far obtained. The most recent statistics show a mortality of but 18.98 per cent., and in Bordeaux of only 11.03 per cent. (Dr. Jonchères, Thèse de Bordeaux, 1895). The duration of the period of impunity conferred by the antitoxin has not been determined but it would appear to be about two or three months.

Tonsillar and Peritonsillar Abscess.

Definition.—By this name we designate an acute inflammation passing on to suppuration either of the tonsil alone, of the cellular tissue which surrounds the tonsil, or of both together. The last case is the most frequent and it is exceptional, if not impossible, for the inflammation to attack one tissue without the other; but as there is in most cases a predominance of the process in one or the other tissue we may practically, following Lasègue, describe the tonsillar and the peritonsillar phlegmon.

Etiology and Pathogenesis.

This is identical with that of catarrhal anginas. These phlegmons occur in young people and especially in those having enlarged tonsils with crypts.

Infection may come from without or more rarely from the blood. The matter accumulated in the crypts and the microbes which are found there normally appear to us to be the most active factors in the production of these phlegmons. A depressed general condition due to a previous disease would favor the growth of these germs. These are usually streptococci, staphylococci, and pneumococci, either singly or associated.

Pathological Anatomy.

During the first stage of the affection there is simply an increase in size of the tonsil, an infiltration of embryonal cells in the submucous layers, an enormous dilatation of the vessels, and an increase in the diameter of the closed follicles. The same inflammatory lesions are present in the peritonsillar tissues. Later we find one or several purulent collections which differ in no particular from collections of pus in general.

Symptoms.

Intratonsillar Abscess (Quinsy).—After a simple tonsillitis has lasted three or four days improvement sets in, the pain on swallowing becomes less marked, the velum palati appears less red, and the general symptoms have disappeared, when suddenly, without any ap-

parent cause or after a new exposure to cold, there is a return of all the symptoms; the fever reappears, the tongue again becomes dry, the appetite fails, and the dysphagia becomes marked; upon examination we now see a red projecting tonsil, greatly increased in size, and a red and œdematous velum and uvula. The tonsil is painful to the touch, much more so than the surrounding parts; there is sometimes a little difficulty in opening the mouth and pressure below the angle of the jaw excites a deep-seated pain; reflex otalgia is not uncommon; an abundant and viscous saliva dribbles from the patient's mouth, for he is unable to swallow anything.

This condition does not very readily pass over into suppuration, and sometimes the symptoms will all disappear within three or four days. At other times, after one or two remissions, the disease returns in all its severity, the general symptoms become aggravated, and we see headache, torticollis, fever, and somnolence; the occurrence of chills betrays the formation of pus. The latter is soon evacuated spontaneously and this, which imparts an infectious odor to the breath, is followed by immediate relief to the patient; within twenty-four hours the pain will have disappeared, the tonsil will have returned to its normal size, and all the general symptoms will have subsided. The pus thus discharged through the mucous membrane of the tonsil is usually small in amount; it may exude in drops at several different times, and the cure is in such cases prolonged. In rare cases, especially when the inflammation has been intense, there is sloughing of a portion of the tonsil, resulting in the formation of a sanious ulcer, which may require two or three weeks to heal. There are also cases in which the abscess has been several weeks in course of development, almost without the patient's knowledge (Cornil), his only complaint being a slight difficulty in swallowing.

Peritonsillar abscess is almost always the result of a tonsillitis which may have been mild or severe. The symptoms are a little stiffness and malaise, headache, a disgust for food, and especially pain and tenderness in the upper portion of the tonsillar region; these are soon followed by an intense chill or by repeated chills, the throat becomes dry and the fever increases. In the course of some days the general symptoms become serious, there is a swelling of the tonsillar region, and we note a unilateral red œdema of the velum palati and uvula, a contraction of the jaw muscles, and an almost complete inability to speak or to swallow. As in the preceding case, there is a dribbling from the mouth of viscous saliva and there is pain at the angle of the jaw, the region is swollen and boggy, and there is a more or less considerable and always painful adenitis.

On the fourth or fifth day after the initial chill the disease termi-

nates by resolution, or more frequently pus collects behind the tonsil and, although the redness in the neighborhood of the abscess is less intense, the pain persists and fluctuation may be made out between the upper third and the lower two-thirds of the anterior pillar. The determination of this fluctuation is, however, painful to the patient and often difficult because of the contraction of the masseters. If the abscess is not interfered with it will usually discharge itself into the mouth at the end of from six to nine days. The opening is ordinarily formed between the tonsil and the anterior pillar, but it is not very rare to see the pus well up from the tonsillar crypts, and sometimes it even forces its way through the anterior pillar. This rupture is followed by a speedy cure; within twenty-four hours the local phenomena as well as the general symptoms disappear almost entirely. If the inflammation has been very intense it will take a little longer to heal, for then the pus usually discharges itself intermittently.

Peritonsillar abscesses have been observed in the course of malignant infectious diseases, such as measles, scarlet fever, or influenza. In such cases they develop slowly and insidiously, produce sloughing of the pharynx, and may cause the death of the patient.

Instead of being unilateral they may be bilateral, either simultaneously or successively, and the course may not be exactly that which we have described as typical.

Complications.

Œdema of the epiglottis is possible, though rare. Hemorrhages from the internal carotid or internal maxillary and thromboses of the jugular veins are exceptional complications. More frequent complications, or rather sequelæ, are hypertrophy of the tonsil or the establishment of fistulous tracts in the substance of the tonsil or of the anterior pillar; the latter is often the fault of the operator who has incised the pillar too high up, leaving a pocket between it and the tonsil.

Diagnosis.

Before the abscess has opened it may be mistaken for a lateropharyngeal phlegmon, for this causes a unilateral bulging of the tonsil toward the median line and also redness of the velum; but the neck is swollen and boggy, and by combined internal and external palpation it is possible to detect fluctuation behind and outside of the tonsil. After the abscess has opened the history of the case will suffice to indicate the nature of the ulceration then present, and to prevent its being confounded with a simple lacunar abscess or a gumma of the tonsil.

Prognosis.

This is usually devoid of gravity, as regards the abscess alone, but it should be guarded because of the possibility of complications.

Treatment.

It is the rule to-day not to wait for the appearance of pus before incising the inflamed tissues, and in this way we often prevent suppuration and shorten very considerably the course of the disease. Therefore as soon as we find a red and inflamed tonsil becoming exquisitely painful and causing œdema of the velum palati and of the uvula, the indication will be to open widely its crypts by means of a sharp hook or a knife with rectangular cutting edge. This reduces the tension of the inflamed part, causes a local bleeding, quickly relieves the pain, and brings about a reduction of the œdema.

If the pus has already been formed in the tonsillar tissues it must be evacuated in the same way. When we have to do with a peritonsillar abscess we should open it through the anterior pillar by a longitudinal incision 2 cm. in length, $\frac{1}{2}$ cm. from the free border. After this incision the parts should be washed frequently with a boric-acid solution, and emollient gargles should be used. In suitable cases the abscess may advantageously be opened with the galvanic knife, for this leaves a gaping orifice through which the cavity will discharge itself completely.

CHRONIC TONSILLAR AFFECTIONS.

Hypertrophy of the Tonsils.

Definition.—A permanent increase in volume of the collection of lymphoid tissue lying between the two pillars of the fauces.

Etiology.

Hypertrophy is most frequent in childhood in lymphatic subjects. Its etiology is the same as that of adenoid vegetations of the nasopharynx and of hypertrophy of the lingual tonsil. Exceptionally the enlargement of the tonsil may be of tubercular or syphilitic origin. Repeated attacks of pharyngitis, even though they may not cause directly hypertrophy of the tonsils, nevertheless contribute to prevent the normal reduction in size of the glands which takes place in adults, and to develop in the interstices of the closed follicles a sclerosis which will bring about induration of the entire gland. The attempt has been made to divide this affection into soft hypertrophy

of childhood and hard hypertrophy of adult life; but this distinction loses much of its value by the fact, which has been repeatedly observed, of large, soft tonsils in individuals over fifteen or thirty years or even in those over forty years of age.

Pathological Anatomy.

In an hypertrophied tonsil the crypts are always deep, and at the surface of the follicular projections, which are more or less enlarged, is a stratified epithelium the layers of which are greatly increased in number. There is, even at the surface, a sort of epidermic transformation of the cells, which, however, we have never observed to go as far as keratinization. On the walls of the crypts the epidermic layers are much less numerous, but there as well as on the surface is a deep limiting layer, very distinct, formed of cuboidal cells. The derma is, in hard tonsils, thickened and formed of a variable thickness of fibrous tissue which sends prolongations into the substance of the gland. In soft tonsils the connective tissue is much less abundant. In both the closed follicles are greatly increased in volume; but while in the hard tonsil the vessels have thick and sclerosed walls, their lumen being consequently narrowed, in the soft tonsils the vessels, which are just as numerous, have almost normal walls or sometimes those walls are scarcely organized.

Symptoms.

Many of the symptoms formerly attributed to hypertrophied tonsils are to-day laid to the account of adenoid vegetations, which are a frequent accompaniment of tonsillar hypertrophy; these symptoms are defective hearing, deformity of the palatine vault and of the upper dental arch, misshapen thorax, a hebetudinous air, etc. Sometimes the enlarged tonsils interfere a little with respiration, but it is seldom that they do not leave between themselves, when the patient is at rest, a sufficient space to assure the passage of the air needed for respiration. The modifications in the timbre of the voice (a nasal intonation, long called the tonsillar voice) really exist, but in order that they may be produced the tonsils must be so large as to interfere by their size with the function of the velum palati. Tonsillar reflexes have been observed, but they are rare.

Upon examination, when the tongue is strongly depressed, we see two tumors, of variable size and often unequal in volume, occupying the triangle formed by the pillars and advancing more or less toward the middle line, that is, toward the uvula, especially when a contraction of the velum occurs. These rounded tumors are more or

less altered in shape according to their attachment, whence we have the various forms of incarcerated, free, pedunculated, and dependent tonsils. The surface is sometimes divided into two or three separate lobes by wide and deep grooves, whence the terms bilobed, trilobed, etc., which have been applied to the hypertrophied tonsils.

It is easy to be misled as to the size of a tonsil upon simple inspection, a free or disengaged tonsil appearing always larger than an incarcerated one, for at the moment of contraction of the velum it is projected far out into the isthmus of the fauces.

The consistence varies greatly, but as a rule the tonsils are soft in children and hard in adults—a rule, however, to which there are many exceptions. The glands at the angle of the jaw are often increased in size, and there is usually hypertrophy at the same time of all the adenoid tissue in the pharynx—Luschka's tonsil, the lingual tonsil, etc.

Complications.

Repeated attacks of tonsillitis are at the same time the cause and the result of hypertrophy of the glands. All infections—either general ones such as tuberculosis, or those that are localized in the pharynx—may enter through the mucous membrane of the crypts; and all the acute and chronic forms of tonsillitis, simple or suppurative, find a favorable occasion for development in hypertrophy of the tonsils.

Diagnosis.

Examination of the patient will be sufficient to exclude the possibility of error. Tumors of the tonsils, syphilitic lesions (chancre, hypertrophied mucous patches) are unilateral and the color of the gland is modified by their presence. We must examine carefully to determine that enlarged vessels or a true angioma are not the cause of a forward projection of the tonsil simulating an hypertrophy. Tonsillar hypertrophy often disappears with age, a sufficient explanation why the condition is less common in adults than in children. Nevertheless it is not very rare to find hypertrophy of the tonsils in adults, as also hypertrophy of the other lymphoid tissues in the pharyngeal region, such as adenoid vegetations and hypertrophy of the lingual tonsil.

Treatment.

The aim of treatment is to cause the disappearance of the whole or the greater part of the lymphoid tissue constituting the hypertrophy. A general tonic medication or the use of iodine fails in most

cases, and local applications are ineffectual, except possibly that of an aqueous solution of resorcin (fifty per cent.) which may bring about a diminution in size of certain soft hypertrophies.

Total ablation has many partisans. It is performed by means of the tonsillotome of Fahnestock or of the galvanic loop, and never now with the probe-pointed bistoury. The tonsillotome and the loop can be used only when the tonsil is projecting and not incarcerated, and the latter has perhaps the fault of removing the whole of the tonsil. The tonsillotome, which is a cutting instrument, ought to be used only in the case of soft tonsils, that is to say, in general, in children up to the age of six or seven years; and it is well in this case, as in others, to attack only one of the glands at a time. The galvanic loop requires a complicated apparatus which is ordinarily in the possession of a specialist only.

Another method consists in the destruction of a certain portion of the tonsillar tissue in such a way as to lead to sclerosis of the balance; this may be accomplished in several ways: (1) By cauterization with the hot iron (thermo-cautery, galvano-cautery), or (2) by discission followed by removal piecemeal.

The thermo-cautery is difficult to manage when operating at the bottom of the throat and especially in patients who are naturally not very docile; it necessitates the employment of a mouth gag, of a tongue depressor, and of guards to protect the internal surface of the cheeks from the heat radiating from the cautery. It has nevertheless the great advantage that it is available by all practitioners. The galvano-cautery is much more easy of application, there is less radiation, and there is also the incontestable advantage that it can be carried cold down to the place which is to be cauterized.

With one or other instrument two or three deep and parallel incisions are to be made into the substance of one tonsil down toward the pedicle. The parts must have been previously anæsthetized by the use of cocaine. Cicatrization takes place in from eight to fifteen days and forms a whitish eschar, often after the production of a simple pseudo-membrane. Little by little, after the cauterization has been practised, the tissue atrophies and the gland shrivels so that its remains are concealed between the two pillars. A second but rarely a third cauterization may be necessary. This method is applicable to all sorts of hypertrophy of the tonsils.

Recently it has been advised to practise discission by means of the sharp hook or a curved probe-pointed bistoury, followed by morcellement by means of the cutting forceps (Ruault). In this way as much may be removed as is desired, no injury is done to the pillars, and the result is immediate. This method is applicable equally

to incarcerated and to pedunculated tonsils. It causes but little pain and is free from danger.

The removal of a tonsil is usually a very simple operation, free from risks, provided the cautions expressed in the previous description of the different methods are observed. It will be prudent to disinfect the proposed field of operation with washes of lemon juice for some days before the removal is attempted, to have the patient dissolve little pieces of ice in the mouth both before and after the operation, not to operate while inflammation is present, and not to remove the entire gland down to the pedicle. In this way we shall stand the best chance of avoiding any complications following the operation.

The most to be dreaded of all the complications is hemorrhage, either primary or secondary; this has, unfortunately, only too often been so abundant as to cause the death of the patient. Primary hemorrhage will in most cases be avoided by not using cutting instruments for indurated tonsils in which the vessels with sclerosed walls are incapable of contracting; if cauterization is practised it should be with a dull-red heat, and the patient should eat only soft and cold food for three or four days after the operation. If in spite of these precautions hemorrhage occurs, it must be arrested by pressure on the bleeding point, by touching it with the cautery at dull heat, by compressing the carotid, or even by ligating this vessel as has been found necessary in several cases.

Antisepsis by means of applications of lemon juice before the operation, the employment of boric-acid lotion, and most careful disinfection of the buccal cavity will prevent simple or diphtheritic inflammation of the stump and œdema of the velum palati, which may extend even to the subglottic region (Bouchacourt).

Caseous (Lacunar) Tonsillitis.

Symptoms.

The functional symptoms experienced by the patient are ordinarily rather vague and but slightly characteristic. There are pricking sensations in this part of the pharynx, some pain on deglutition, and the expulsion in greater or less quantity of the very characteristic grayish-white, caseous, and extremely fetid concretions. The patient often complains of the fetid taste in the mouth from these concretions, which give him also a bad breath. Not infrequently the expulsion of some of these concretions will be followed by a temporary amelioration of the local troubles.

On examination the tonsils are found to be enlarged or atrophied.

It is not uncommon, upon depressing the tongue and causing the tonsils to project, to see upon their surface, usually on the upper part, this characteristic grayish material. In other cases the lacunæ, which are often quite deep, can be evacuated of their secretion only by means of the stylet or a dull curette. At first sight this matter resembles a pultaceous coating, but upon making a little pressure on the tonsil we see that we have a concretion and not an exudate. The collections may in rare cases become encrusted with calcareous matter, so as to form true amygdaloliths, tonsillar calculi, the symptoms of which will not differ from those of caseous tonsillitis.

Treatment.

The proper treatment consists in opening freely the tonsillar crypts, by means of a sharp hook or elbowed bistoury. In the hypertrophic forms, instead of incision, we may perform morcellement by means of Ruault's forceps. In this way we break down the septa between the lacunæ and at the same time relieve the patient of his hypertrophy.

Mycosis of the Tonsils.

This affection, which was first studied by Fraenkel and a little later by Gumbiner, Heryng, Bayer and others, is characterized by the presence on the lingual or buccal tonsils, of collections of leptothrix buccalis.

The functional symptoms are of the most varied character and resemble quite closely those of chronic lacunar tonsillitis, of which we have just been speaking. Often they are but slightly marked and may pass unperceived. At other times, on the contrary, the patient has a pasty feeling in the mouth, and the tongue feels rasped, as if he had been eating raw artichokes.

Inspection shows us little yellowish projections from the tonsils, forming a sort of hairy growth, disposed in bundles, projecting one to several millimetres above the surface. They are very adherent to the tissues which support them, have a horny consistence, and are removed with difficulty.

In order to cure the affection we often have to resort to a true epilation. At other times this result may be obtained by lotions of chloride of zinc, 1:15, and then of a solution of iodine, 1:5. Wagnier, of Lille, recommends chromic acid as very efficacious, while others employ the galvanic knife. If one of these methods does not succeed we may resort to another, remembering always that the affection is local, although frequently obstinate, and that it is always possible

with a little patience on the part of both physician and patient to obtain a definite cure.

Tuberculosis of the Tonsils.

Etiology.

The tonsils, like the other organs, may be invaded primarily or secondarily by the tubercle bacilli; the second method is by far the more frequent but is usually observed only in cases of advanced tuberculosis of the larynx or lungs. Certain authors (Dieulafoy) have recently advanced the opinion that most cases of hypertrophy of the tonsils are really latent tuberculosis, but this opinion is hardly tenable, for it is supported by no facts whatever.

Symptoms.

Lupus.—Lupus of the tonsils is rare; it arises by extension of a similar affection of the pharynx, of the pillars, or of the velum palati and presents the same appearances here as in those regions. The tonsil is purplish, granular, and slightly increased in size through infiltration of its mucous membrane; it is indistinguishable from the pillar of the fauces and may, like it, present soft, reddish ulcerations with slightly projecting and illy defined borders; little by little the tonsil decreases in size and finally there is left simply a whitish cicatricial tissue, indistinguishable from that of the neighboring parts. The affection is almost wholly painless; it is not accompanied by adenopathy, and as it never appears as a primary affection it has but very slight importance and this only as regards its results.

Tuberculous Ulceration.—This is much more frequent than lupus; it is always preceded by a stage of miliary infiltration of the surface of the tonsil. In a case of advanced tuberculosis this period of infiltration is ordinarily announced by symptoms of an intensely acute angina. The invaded tonsil is swollen, red, and shining and on its surface are three or four little whitish projecting points, true miliary tubercles, which advance very rapidly, sometimes in two or three days, to the stage of ulceration. These little losses of substance unite to form a single ulcer which also increases a little in depth as well. When once it is formed it is deep, uneven, and with irregular borders; the tonsil upon which it is located is red, the surface of the ulcer is grayish and covered with a pultaceous coating, and its borders are surrounded by fresh tuberculous granulations. There are sore throat, an abundant salivation, engorgement, and sometimes suppuration of the cervical glands on the same side.

Besides this acute form of tuberculous ulceration of the tonsil,

there is another slower, somewhat chronic form, the symptoms of which are much less pronounced. There is slight pain, localized on one tonsil, and inspection shows on the surface of this gland a granular, slightly elevated patch, the surface of which rapidly undergoes superficial erosion, the first stage of the chronic ulceration. Soon this ulceration increases, the uneven surface is dotted with red and gray points, and it may vegetate and become covered with papilliform growths similar to exuberant granulations. These growths may themselves become sufficiently voluminous to suggest a polyp of the tonsil, one to several centimetres in length. During a variable period of time, several months, the ulceration makes little progress and the functional symptoms are but slightly marked. Then, under the influence of an acute exacerbation of the disease in the lungs or in the larynx, the lesions may become acute, disseminated granulations appear at the periphery of the ulcer, and the general appearance recalls that of the acute form. This chronic ulceration constitutes a sort of transition between lupus and the acute form of tuberculous ulceration. Ordinarily the pharynx, the lungs, and the larynx are involved at the same time or their lesions precede those of the tonsil and mask them.

Latent Tuberculosis.—Occasionally—once in forty times according to Cornil, but less frequently in my experience—upon cutting hypertrophied tonsils which had presented nothing suspicious, we find tuberculous deposits, with caseous points, giant cells, and Koch's bacilli. The mucous membrane is not ulcerated, the patient appears to be healthy, and there is no history of any tubercular trouble. The lesion occupies usually one of the closed follicles, the tissue around which is normal or at most there are a few points of congestion. We have no clinical reports of cases of this kind, which may be said to belong purely to the pathological laboratory. On the other hand, we know that there are upon the surface of the mucous membrane and within the crypts of healthy tonsils bacilli of various sorts, including the tubercle bacillus. May some of these bacilli pass through the mucous membrane? We may assume that they do, but we cannot prove that there is not in some other part of the organism one or several identical nodules which will heal, or that this concealed tuberculous nodule may not have been surrounded by a zone of sclerosis and have itself become cured. We can readily understand that the bacilli of this primary centre may invade the neighboring glands, the lungs, the joints, etc., and consequently that an hypertrophy of the tonsil, which was supposed to be simple, may contain in itself germs which will cause more or less rapid infection of the individual.

Course, Duration, Termination.

The course of lupus is slow, the disease progressing by several successive exacerbations. When localized in the pharynx or in the tonsil the affection is not incompatible with a condition of apparently excellent general health.

The progress of tuberculous ulceration is more rapid (six months or a year for the chronic form, some days or weeks only for the acute form), and the patient is carried away, not by his tonsillar lesion, but by tuberculosis of the larynx or lungs.

Our data for the study of larval tuberculosis of the tonsils are as yet very incomplete, and it is not impossible that certain chronic inflammations of the gland will be found to belong to this category. It will probably be found also that the affection, being a local tuberculosis, is capable either of being cured or of infecting the organism.

Prognosis.

The prognosis rests upon what has been already said. Tuberculosis is always a grave affection, but there is a great difference between lupus and acute tuberculous ulceration of the tonsil; a local affection is always susceptible of cure, one which keeps step with the lesions in other organs which have given it birth is an announcement of speedy dissolution.

Pathological Anatomy.

The following are the lesions which I have found in examining the borders of a tuberculous ulcer. The closed follicles of the tonsil are no longer recognizable, their place being taken by an infiltration of round cells the nuclei of which take a bright stain with picrocarmine. The epithelium is much thickened in the non-ulcerated portions. There is also a marked dilatation of the blood-vessels of the parts, with thickening of the walls of these vessels and an infiltration of these walls by lymphatic cells.

In several points are seen a rather large number of giant cells the nuclei of which take a bright stain, and which are surrounded by embryonic cells. Finally we notice near the free border of the ulcer little caseous masses recognizable by their uniform pink color and by the absence of a nucleus. As we recede from the border of the ulcer the tonsillar tissue regains little by little its normal appearance, although there are still found giant cells in several places, each one surrounded with an inflammatory zone.

The tubercle bacilli are few in number, and are met with especially in the inflamed areas surrounding the giant cells.

Diagnosis.

Simple ulcer has been described above; it could scarcely be mistaken for a tuberculous lesion. Syphilitic ulceration (chancres, mucous patches, gummata) will be studied in the following section, and the diagnostic points will be considered there. Pseudo-membranous ulceration (herpes, aphthæ) is much more superficial and smaller in extent; the lesions develop in an acute form and have the appearance of little cup-shaped depressions surrounded by swollen mucous membrane. The presence of herpes, aphthæ, or muguet on the buccal mucous membrane will remove all doubt as to the nature of the lesion. The lesions called ulcero-membranous exist in connection with similar lesions in the mouth; they bleed easily, are superficial and irregular and at the same time very painful; they yield rapidly to the internal use of chlorate of potassium; and still another symptom which would lead us to think of this lesion is the especially fetid breath which is associated with it. The ulcers seen in connection with eruptive fevers (typhoid, scarlatina, small-pox) are but an epiphenomenon in the evolution of these fevers the existence of which will sufficiently explain the nature of the tonsillar lesion. Ulcerative epithelioma has an indurated base; it is unilateral, the tonsil is projecting, the pillars are immobile, the breath is fetid, and adenopathy comes on rapidly; the general condition rapidly deteriorates, the patient becomes weak, and there is a speedy extension of the disease to the neighboring parts.

Treatment.

For the ulcers of lupus applications of chloride of zinc, 1 :10, or of lactic acid have been highly recommended. Phenol sulphoricin (Ruault) or carbolized glycerin (Moure) in the strength of 1:10, 1:5, and then 1:3 will rapidly bring about cicatrization, especially if the applications have been preceded by the use of the curette.

If there are polypoid growths they should be removed and their points of insertion should be cauterized.

Chronic tuberculous ulcerations are amenable to the same treatment as lupus ulcers; even the acute ulcers may be brought to cicatrize by the application of these agents, but that will not prevent the patient from being carried away by the laryngeal or pulmonary trouble. Applications of a solution of hydrochlorate of cocaine will relieve the pain temporarily and will enable the patients to take a sufficient amount of nourishment, and this is an enormous advantage in a disease of such a nature.

Syphilis of the Tonsils.

All the varieties of syphilitic lesions may be observed in the tonsil; we shall study in order the primary, secondary, and tertiary lesions.

Chancre.

The initial lesion of syphilis is developed in the tonsil more often than was formerly supposed. It is the result of direct inoculation (*coitus ab ore*, mucous patches on the breast of the nurse) or of indirect inoculation through the indiscriminate use of cups, forks, spoons, etc. The existence of the tonsillar crypts and of the erosions which are so frequent on the surface of the tonsil singularly favors the occurrence of the initial lesion on this gland. Tonsillar chancre is in most cases unilateral, but sometimes the lesions occupy both glands. It may present, according to Fournier, under four different aspects, viz., anginous, diphtheroid, eroded, and ulcerative.

Before speaking of each of these forms we may discuss the characters which are common to them all. The tonsil is enlarged, extending toward the middle line even up to the edge of the uvula. Its substance is indurated, its mucous membrane reddened, and it is painful to the touch. On the same side as the lesion, behind the angle of the jaw, there are one or several enlarged and painless glands, but slightly movable and surrounded by a collection of small glands. The adenopathy is considerable enough to cause an external swelling that is distinctly appreciable to the naked eye. The skin covering the enlarged glands is of normal color and mobility.

The presence of the chancre is sometimes announced by a sharp inflammatory reaction in the throat, accompanied by intense dysphagia, painful adenitis, and fever. Instead of yielding in three or four days the pain persists; the tonsil, which was at first only red, now swells, and on its surface appear pultaceous deposits; the adenitis increases, and we now see that we are in the presence of a much more serious affection than a simple angina, such as we thought at first from the appearance of the tonsil and neighboring parts. We ought always to think of the possibility of such a lesion whenever a sore throat persists a long time, and we should examine the external surface of the body with great care to see whether there are any signs on the skin which would confirm our suspicions of a syphilitic infection.

We sometimes see a chancre under the form of slight tonsillitis in which the gland is increased in size, red, and covered with a pseudo-membranous exudate, which would suggest upon superficial exami-

nation a diphtheritic tonsillitis. But if we take away the false membrane we shall see a small erosion seated upon a very indurated base; the cervical glands are much swollen, the pseudo-membranous exudate does not pass beyond the limits of the tonsil, the pain is almost *nil*, the affection remains unilateral, and its duration is from two to three weeks. There is an ulceration on a projecting portion of the tonsil, the borders are indurated, the tonsil increases daily in size, the cervical adenitis increases, and sometimes even before the disappearance of this first erosion a mucous patch appears on its site.

It quite frequently happens that tonsillar chancre appears under the form of true ulceration, involving the entire mucous membrane and subjacent tissue; the surface of the ulcer is grayish and covered with a pultaceous deposit, its borders are irregular and indurated, the gland is more or less increased in size, and the loss of substance through ulceration appears to be considerable; the adenopathy, which is never absent, will put us in the way of a correct diagnosis.

A tonsillar chancre is sometimes confounded with an epithelioma; there are the same induration and the same adenitis, but the two affections are very unlike in their progress; in the case of chancre the adenopathy appears much earlier, and the secondary lesions quickly supervene, removing all doubts as to the nature of the affection.

Secondary Lesions.

Some authors have treated of a syphilitic angina corresponding to the cutaneous roseola and affecting especially the velum palati and the tonsils, but this view is far from being universally accepted. The secondary lesions which are recognized by all to be such are tonsillar hypertrophy and the mucous patch.

The hypertrophy is bilateral, almost painless, and in no way to be distinguished externally from simple hypertrophy. The two tonsils come together in the median line and give to the voice a very characteristic nasal intonation. The mucous membrane sometimes ulcerates at the points where the two glands are in contact, and mucous patches may also develop on the surface of the tonsils. This hypertrophy is accompanied by cervical adenitis; sometimes it disappears entirely in a few days and sometimes it passes into a chronic condition; it predisposes to inflammatory affections of the pharynx, and sometimes even to abscess.

The tonsil is a favorite seat for the mucous patch. The latter appears under various forms, such as the eroded, the papulo-eroded, the papulo-hypertrophic, and the ulcerative; the first and the third of these are the most usual forms.

The eroded mucous patch has the appearance of a round or oval ulcer, distinctly limited, with a moist grayish surface, edges little or not at all projecting, and surrounded by an excessively fine red line. The papulo-hypertrophic form is slightly polypoid in appearance; it is pinkish in color and is raised a little above the surrounding red and slightly œdematous tissue; it occupies projecting portions of the gland. There is in this, as in the first form, rather severe pain caused by the passage of food. As is the case with mucous patches in other parts of the mouth, those of the tonsil are seen most frequently in smokers, drinkers, and in general in those who habitually use any substance irritating to the throat. I have sometimes seen in these cases veritable hypertrophic patches, having a strong resemblance to diffuse papillomata.

The secondary lesions of the tonsils may last several months and may recur at intervals for several years. They often constitute the first palpable sign of syphilitic infection, the initial lesion having passed unperceived. They frequently coexist with similar lesions on the other mucous membranes and like them are amenable to general and local specific treatment. I usually prescibe in such cases the following:

Biniodide of mercury,	0.15
Iodide of potassium,	15.0
Anisette,	50.0
Distilled water,	275.0

Dose: Dessertspoonful in milk morning and evening.

Local applications of pure tincture of iodine or of the acid nitrate of mercury have always given excellent results in my hands. The patient should be cautioned not to smoke nor to drink any alcoholic or other irritating beverage, for we know by experience that treatment is often of no avail if these precautions are not followed.

Gumma.

The only tertiary lesions encountered in the tonsil are gummata and the ulcerations which result from them. It is seldom that we have the opportunity to observe a gumma of the tonsil at its inception. In such cases the gland is slightly enlarged, painless, and ulcerates rapidly; it is on account of this that we so seldom have the opportunity of witnessing the lesion before ulceration has taken place. The ulcerated gumma appears under the form of a clean-cut loss of substance covered with a pultaceous yellowish exudate, beneath which is a sanious, reddish, and fungating surface. The characteristic of the gummatous ulceration is that it does not remain confined to the central area of the tonsil, but, on the contrary, appears on its edges

and very rapidly invades the pillars of the fauces, the velum, the pharynx, or the base of the tongue, extending constantly in area and depth. Tertiary syphilis of this region, therefore, when untreated, produces extensive destruction of tissue, leaving behind it characteristic cicatricial synechiæ. The edges of the syphilitic ulcer are always more or less red, inflamed, and infiltrated—appearances never seen in simple ulceration.

The ulceration takes place after softening of the gumma, and this process of softening extends to the neighboring parts, sometimes even the vertebral artery (Mackenzie). Before ulceration has taken place the gumma constitutes a fluctuating tumor and the mucous membrane covering it is injected and of a purplish-red color. There is so little tendency to cicatrization in the case of a gummy ulcer that Morell Mackenzie has well said, when a suspicious ulceration persists obstinately for four or five weeks in spite of internal remedies (specific treatment excepted) and local applications, such as nitrate of silver, acid nitrate of mercury, etc., we may almost positively assert the specific nature of the affection.

The preceding description relieves us from dwelling at length upon the diagnosis. We need only add to what has been already said, that gummy ulceration is never accompanied by adenopathy, a fact which will serve to differentiate it from chancre, epithelioma, or even tuberculosis.

The treatment is the same as that for the secondary lesion, the dose of the iodide of potassium may be slightly increased, and we must cleanse the surface of the ulcer carefully before making any local application, such as tincture of iodine, chloride of zinc, or the like. Under this treatment cicatrization proceeds rapidly in the tonsil, and the pillars of the fauces soon resume their normal size and appearance. In cases of long standing, however, we shall almost always see consecutive cicatricial synechiæ.

Foreign Bodies—Calculi.

We have already enumerated in the article on diseases of the pharynx the various foreign bodies which may become arrested in the isthmus of the fauces. The tonsils, in consequence of their often considerable size, especially in children, frequently arrest objects, such as fish bones, pins, pieces of bone, etc., which give rise to precisely the same symptoms and results as foreign bodies of the pharyngeal wall or base of the tongue. In this section we shall consider only those foreign bodies which are not found in other parts of the fauces, viz., calculi.

Etiology, Pathogenesis.

We see the mucous membrane on the surface of the tonsils dipping down into cavities which divide the gland into lobes and lobules; these depressions or crypts are more or less deep and may become distended by epithelial products, mucus, particles of food, etc., whenever the edges of the crypt become glued together or whenever they become rigid so as to be incapable of separation.

Calculi of the tonsils, which are not at all common, are met with especially in individuals with large tonsils having deep lacunæ. The acinous glands on the surface of the mucous membrane may have their secretion increased, and the crypt itself becomes the seat of a chemical and microbic change of the saliva accumulating there, of the dental tartar which is deposited there, of particles of food, and of the cells desquamated from its walls. One of these foreign bodies may become the centre of a calculus, the mineral salts of which are derived from the saliva.

Composition.

The following is the result of the chemical analysis of a calculus which I obtained from a tonsil:

Phosphate of magnesium,	4.15 per cent.
Phosphate of calcium,	66.80 "
Carbonate of calcium,	4.10 "
Organic matters,	16.25 "
Water,	8.70 "

In many other cases nearly identical results have been obtained. The histological and chemical examination of these calculi shows their origin to be that above indicated.

Symptoms.

A calculus may exist for a long time without giving rise to any symptoms, the first intimation of its presence being its expulsion. In other cases there is a disagreeable sensation, a little pricking on one side, or repeated attacks of tonsillitis, leading gradually to sclerotic hypertrophy of the organ. In other cases there is more or less irritation leading to a croupy cough, vomiting, or redness of the mucous membrane. These phenomena may exist for many years without their true cause being discovered. Sometimes a tonsillar abscess will lead to the discovery of the cause of the symptoms, sometimes the attention of the physician is drawn to the tonsil by a series of abscesses occurring without known cause. In some cases the calculus may be seen to project from a crypt, or palpation may reveal

the presence of an induration in the tonsil existing at the seat of the buried calculus. Exploration of the crypts by means of a stylet will reveal the presence of the foreign body or bodies (for there may be several).

Diagnosis.

The sensation as of a pebble obtained on exploration with the stylet will prevent any mistake in diagnosis. Nevertheless, as we may not suspect the presence of the calculus there will be danger of mistaking the induration, the hypertrophy, and the nodular enlargement of a tonsil containing a calculus for a cancer; the adenitis at the angle of the jaw resulting from the inflammation of the tissues around the crypt containing the calculus may add another element of confusion. The mistake has been made. Simple hypertrophy is ordinarily bilateral and there is rarely such an induration as exists with calculus. The caseous masses of lacunar tonsillitis have not the consistence of a calculus and may be evacuated by simple pressure; the same may be said of the whitish and filamentous masses of mycosis. It is seldom possible to determine whether one or several calculi are present in the tonsil.

Prognosis.

This is favorable, although we must remember that a calculus may lead to repeated attacks of tonsillitis, resulting in hypertrophy of the gland with all its consequences, and possibly in abscesses which may in their turn become the origin of peritonsillar phlegmons. Hence intervention is necessary.

Treatment.

The only rational treatment consists in extraction of the calculus. The crypt may be opened widely by means of the cutting hook, when it will be easy to seize the calculus with forceps of various shapes. The reproduction of the calculus may be prevented by opening all the crypts and biting away the surface of the tonsil by means of cutting forceps. The ablation of the tonsils by the tonsillotome or galvanic loop is inadvisable here, but igni- or galvano-puncture may be of service.

Tumors.

Tumors of the tonsil may be benign, malign, or mixed. The latter so nearly resemble malignant tumors that they may well be treated in the same section with them. We shall study successively the benign tumors, which include pseudo-polypous hypertrophy

(papilloma), fibroma, and hydatid cyst; and the malignant tumors, including sarcoma, lymphadenoma, and lymphosarcoma, and scirrhous, encephaloid, and epithelial cancer. Simple and syphilitic hypertrophy, vegetating mucous patches, tuberculous, gummatous, and calculous tumors have already been discussed, so we shall not consider them here.

Pseudo-Polypous Hypertrophy.

There is little to say of this tumor. It may result from the unequal development of one tonsillar lobe, which gradually acquires a pedicle and then hangs from its point of attachment like a polypus. The glandular tissue constituting this false polyp is unchanged histologically, but this does not prevent it from giving rise to the same functional symptoms as other tumors of the gland, viz., difficulty of swallowing, more or less pronounced interference with respiration, nausea, and often vomiting excited by the contact of the tumor with the base of the tongue.

In pseudo-polypous hypertrophy the tissue is of the normal appearance and consistence of the tonsil. Instead of one lobe there may be two or three, but in such case the pedicle is smaller and the tumors constitute really an hypertrophied tonsil with deep sulci. Pseudo-polypous hypertrophy, like simple hypertrophy, predisposes to inflammatory attacks.

The hypertrophic outgrowth is easily removed by the galvano-cautery, thermo-cautery, or a cutting instrument.

Fibromata and Fibrous Polypi.

Fibrous polypi sometimes arise, though rarely, at the expense of the submucous tissue of the tonsil. The presence of the tumor is revealed by a nasal intonation, snoring during sleep, and attacks of suffocation. The tonsil is more or less enlarged and the tumor may obstruct the isthmus of the fauces, the diseased tonsil extending nearly to its fellow on the opposite side. The tumor is covered by the normal mucous membrane of the tonsil; its surface is smooth, its consistence hard and elastic, giving sometimes a sensation as of fluctuation; it is but slightly movable. There is no pain, either spontaneous or excited by pressure; there is no adenopathy, and dysphagia is absent. Sometimes solid or liquid food is rejected through the nose. When the tumor is not excessively large, respiration through the nostrils is not obstructed. The general condition of the patient is usually excellent.

On section the tumor is seen to be formed of fibrous tissue, arising in all probability from the blood-vessels, as seems to be shown by

the concentric zones of fibrous tissue surrounding and forming the walls of the blood-vessels. Here and there may occasionally be seen cysts with mucous contents, as also islets of tonsillar tissue, which appears to be normal as regards its framework but from the meshes of which the lymphatic cells have often disappeared.

These tumors are usually quite easily enucleated, and they do not return after removal. It may be necessary to divide the anterior pillar of the fauces in order to permit of their extraction, which may be done by means of the finger, the bistoury, or, better still, the galvanic loop. Hemorrhage is not to be feared.

Hydatid Cysts.

Hydatid cyst of the tonsil appears in the form of a smooth, rounded, fluctuating tumor of slow growth and painless; the diagnosis is often made only upon opening the cyst. Cauterization of the interior of the sac after incision or total extirpation is the appropriate treatment for tumors of this class.

From what has just been said it may be seen that tumors of the three classes above described cause trouble only by their size. Nevertheless we must remember that it is not very uncommon to see a tumor which is primarily benign become infiltrated with embryonal elements and assume little by little the character of a sarcoma or of an epithelioma. The removal of such tumors is therefore always indicated. This may best be accomplished through the mouth.

Malignant Tumors.

These may be primary or, more rarely, secondary. We need not occupy ourselves with the consideration of secondary tumors, merely remarking that they are ordinarily an extension from tumors of the tongue or of the pillars of the fauces.

Pathological Anatomy.

We find in the tonsil pure sarcomata, lymphadenomata, and lymphosarcomata (the two latter being characterized by the presence of a delicate reticulum filled with sarcomatous cells), epitheliomata and carcinomata, which differ but little one from the other in their microscopical characteristics. The carcinomata have more connective tissue than the epitheliomata, epithelioid cells predominating in the latter.

Etiology.

We know practically little of the cause of these tumors. Sarcomata appear to be more frequent in young subjects, epitheliomata and

carcinomata appearing in adult life. Neither tobacco nor alcohol seems to have any influence in the etiology of malignant neoplasms.

Symptoms.

The first phenomenon which attracts the attention of the patient is sometimes a difficulty in deglutition; pain in the tonsil, radiating to the gums, in the neck, and in the head; a dryness of the throat, or else an increased salivation; sometimes there is pain in the ear, and finally there is more or less marked cervical adenopathy. The difficulty of swallowing and the pain in the ear gradually increase and there is also pain upon pressure behind the angle of the jaw.

The voice becomes nasal and indistinct and the patient has more and more difficulty in making himself understood. An abundant salivation, as above stated, may be present, causing him to expectorate constantly, and deglutition becomes more and more painful as the pillars are invaded and immobilized by the tumor. There is sometimes a little expectoration of blood and the breath is extremely fetid.

If we are able to examine the throat, which is not always easy because of the stiffness of the jaws and the immobilization of the base of the tongue, as well as by reason of the pain caused by movement of this organ, we note an appearance which varies according to the nature of the tumor.

Sarcoma appears under the form of a hard, nodular tumor of variable consistence, the growth of which is rapid, the seat unilateral, and which, even before there is any adenopathy, may send out prolongations above toward the Eustachian tube, which it may obstruct, or downwards towards the entrance of the larynx, which it may narrow. The tumor is covered with a reddish-purple mucous membrane. It has not had time usually to ulcerate before being discovered, for the lancinating pains which are often present draw the attention of the patient to his tumor and lead him to clamor for its removal.

Lymphadenoma or lymphosarcoma (it is impossible to differentiate the two tumors histologically) is usually of quite rapid growth. It occurs as a painless tumor which soon interferes with deglutition and respiration. Frequently the two tonsils are simultaneously or successively attacked, forming tumors which fill the isthmus of the fauces, ascend into the naso-pharynx, and descend toward the lower portion of the pharynx. These tumors, which are never painful except sometimes when the two meet and ulcerate at their points of contact, may, however, compress the lips of the Eustachian tube, causing violent otalgia and sometimes exciting suppurative otitis, as we have observed recently in a case of which we may here give the outlines, since the history is a typical one of this affection.

A woman, 40 years of age, noticed six months ago a little difficulty in deglutition, a nasal intonation of the voice, and occasionally a regurgitation of liquids through the nose. Upon looking at her throat in the glass she saw that the right tonsil was greatly enlarged. At this time there were but two or three small glands to be felt on the right side of the neck. The physician whom she consulted removed the diseased tonsil; this was followed by an obstinate hemorrhage which continued for three days. Relief followed the operation during a period of three weeks. However, the cervical glands, which had before been scarcely visible, began now to increase rapidly in size, although they were painless and distinct one from the other. Then the tonsil of the other side began to enlarge and increased very rapidly, at the same time that a large collection of glands became noticeable on the left side of the neck. Within a short time a very intense otalgia was felt and a suppurating otitis appeared from which the patient still suffers. The tonsil, which is the size of an orange, occupies the entire isthmus of the fauces, interfering not only with deglutition, but also somewhat with respiration. The tumor is not ulcerated. The glandular tumefaction in the neck is enormous on each side. There has been no return of the disease on the side operated upon, but the patient is emaciating from day to day, is exceedingly pale and weak. We have here evidently a case of leucocythæmic adenia which will soon cause the death of the patient.

Under the microscope lymphadenoma of the tonsil appears in the form of a delicate reticulum filled with round lymphatic cells.

Carcinoma.

This appears under one of two forms, either tonsillar hypertrophy with ulceration or a destructive ulceration of the gland. It is only in exceptional cases that the neighboring parts, especially the anterior pillars, are not infiltrated and invaded by the neoplasm. When the cancer is hypertrophic it has the appearance of a mushroom, the head of which is formed of little nodules looking like the centre of a cauliflower. When ulceration exists the borders are irregular, swollen, and hard. Adenopathy appears early and extends to all the cervical region. Acute inflammation of the glands may take place, leading to phlegmonous adenitis, the walls of the abscess often becoming themselves the seat of cancerous infiltration. This adenopathy bears no relation to the size of the original tumor. Cancer of the tonsil is almost always accompanied by violent otalgia.

The general condition rapidly deteriorates, for the patient suffers much pain, is poorly nourished, excretes an excessive amount of saliva, and swallows much of the ichorous discharge from the tumor. The tumor may become generalized, and very frequently it extends to the neighboring parts, the velum palati, root of the tongue, nasopharynx, vertebral column, base of the skull, parotid gland, and the

neighboring great vessels in the neck; the involvement of the latter is often a cause of severe hemorrhage.

The patient dies of cachexia, erysipelas, hemorrhage, tuberculosis, œdema of the glottis, or phlegmasia alba dolens. Sudden death is frequent.

Diagnosis.

In any case of doubt the trial of specific treatment will prevent confusion with chancre, secondary syphilitic hypertrophy, or gummatous ulceration. Tuberculous growths and ulcers advance more slowly and are accompanied by little or no adenitis, and the patient usually presents other symptoms pointing to the nature of the disease. A foreign body may be a source of error, but it is only necessary to remember the possibility of such an occurrence in order to avoid the mistake. The other malignant tumors may readily be differentiated by noting the characteristics above mentioned.

Treatment.

Some tumors should not be touched; these are the ones that are already accompanied by an adherent or multiple adenopathy, that have passed beyond the limits of the tonsil and of the velum palati, or that have already induced a cachectic state. Leucocythæmic lymphadenomata should also be let alone.

Whenever the tumor has passed beyond the limits of the tonsil and is accompanied by slight and movable adenopathy we must remove it through an external incision. It is often prudent to perform a preliminary tracheotomy, to tie the external carotid, and to remove the tumor either through an enlargement of the normal buccal opening or by means of a temporary or permanent resection of the lower jaw. Other operations are the prolongation of an incision from the labial commissure, meeting the incision made for ligation of the external carotid, or finally, we may make a horizontal subthyroid pharyngotomy. Once the tumor is reached it may be removed piecemeal by means of Ruault's cutting forceps, of the bistoury, of the thermocautery, or, better, of the galvanic loop.

Malignant tumors of the tonsil frequently return after removal and therefore an early operation is advisable, as giving a better chance of a permanent cure.

DISEASES OF THE LARYNX.

BY

FRANCKE H. BOSWORTH,

NEW YORK.

DISEASES OF THE LARYNX.

GENERAL CONSIDERATIONS.

Nature, in carrying out her purpose to endow certain animals with the power of phonation, availed herself of the current of respired air, which, by a very simple valve-like mechanism placed near the outer exit of this current, is thrown into vibration. In this manner sound waves are produced. These are subsequently converted into articulate language by the movements of the soft palate, tongue, and lips; intelligent articulation, however, belongs probably to the human species alone.

The phonatory apparatus we call the larynx, but in order that the respiratory act may in no degree be interfered with by this function, we find the larynx also endowed with certain movements which are called the respiratory function, viz., that function by which the phonatory valves, or vocal cords, are widely opened by involuntary muscular action during the act of inspiration.

The larynx consists practically of such modifications of the upper rings of the trachea as shall fit it for the performance of the special functions of respiration and phonation which it is designed to subserve. This modification is accomplished by expanding the tracheal rings as it were and changing their shape so that there may be inserted a vocal apparatus which shall at the same time admit of the ingress and egress of the respiratory current of air.

Our present consideration demands only the most cursory reference to the anatomy of this organ. Immediately above the upper ring of the trachea there is placed the cricoid cartilage, so called from its resemblance to a seal ring. Above this in front we have the broad thyroid or shield-like cartilage protecting the laryngeal cavity anteriorly by its resisting consistence and box-like contour. Surmounting the upper surface of the broad expanded posterior portion of the cricoid we have the two arytenoid cartilages, small triangular pyramids attached by articular facets. Attached to the receding angle of the thyroid cartilage above we have the flat leaf-shaped cartilage, the epiglottis, whose narrow attachment to the receding angle of the thyroid gives wide latitude of motion. In addition to these cartilages, we

have the two small cartilages of Santorini immediately over the apex of each arytenoid cartilage, and the cartilages of Wrisberg—two staff-like cartilages which are found in the folds of mucous membrane stretching from the arytenoid cartilage on each side to the epiglottis; these, however, possess an anatomical rather than a practical interest.

The essential feature of this structure, and the one for which it was especially designed, is to give a site for the location of the vocal cords. These are two narrow bands of fibrous tissue which are stretched from the anterior angle of the arytenoid cartilages to the receding angle of the thyroid, their anterior insertion being fixed while their posterior insertion into the arytenoids admits of considerable freedom of motion, the outward movement being called abduction and the inward movement adduction.

The only remaining structures of the larynx which are of interest are the mucous membrane which lines it, and the muscles which act upon the movable cartilages. The mucous membrane, like that of all portions of the respiratory tract, is of the columnar-ciliated variety. We describe only those muscles which are of practical interest. The posterior crico-arytenoids, one on either side, pass from the posterior surface of the cricoid ring to the outer angle of the base of the arytenoid cartilage, their action in contraction being to rotate the anterior angle of the cricoid cartilage outwards, thus separating the vocal cords. The lateral crico-arytenoids passing from the outer angle of each arytenoid forwards to the inner surface of the cricoid ring, directly antagonize the former muscles, rotating the arytenoid cartilages inwards, thus closing the cords. This action becomes of exceeding importance in certain diseased conditions, and is well illustrated in Figs. 37 and 38. The arytenoid muscle passes from the posterior face of one arytenoid to that of the other, thus drawing the cartilages together and aiding to close the glottis. Stretched along the under surface of each vocal cord we have the thyroid-arytenoid muscles composed of different sets of muscular fibres whose action is in contraction to render tense the vocal cords, thus regulating the pitch of the voice, a function which is to a certain extent aided by a muscle on the anterior surface of the cartilaginous framework of the larynx, the crico-thyroid, which draws the thyroid down upon the cricoid, thus rendering it fixed.

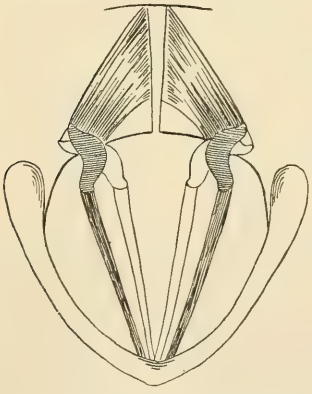


FIG. 37.—The Glottis-Opening Action of the Posterior Crico-Arytenoid Muscles, shown diagrammatically.

The arterial supply is derived from the superior and inferior thyroid arteries, the former being a branch of the external carotid, while the latter has its origin in the thyroid axis, their ultimate distribution forming the superior, inferior, and posterior laryngeal arteries. The larynx derives its motor and its sensory innervation from the superior and inferior laryngeal nerves. The former is a branch of the pneumogastric and supplies general sensation to the mucous membrane lining the larynx, and motor innervation to the crico-thyroid muscle. The inferior, or as it is generally called the recurrent, laryngeal nerve supplies the remaining muscles of the larynx with their motor innervation.

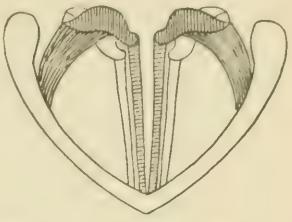


FIG. 38.—The Glottis-Closing Action of the Lateral Crico-Arytenoid Muscles, shown diagrammatically.

This brief reference to the anatomy of the larynx is, I think, quite sufficient to enable us to attain to a thoroughly intelligent comprehension of the function which this organ is designed to subserve in the economy, viz., that relating to respiration and phonation. The contour of the interior of the larynx is such that the current of air in expiration easily makes its escape by forcing the vocal cords asunder as it were, while the ingoing current, striking as it does on the broad flat surface of the vocal bands, would act to throw them together in a manner not unlike the mitral valves of the heart, were it not for the fact that nature provides muscular action by which the glottis is opened with every act of inspiration; this is accomplished by the posterior crico-arytenoid muscles, which, drawing the outer angle of the arytenoid cartilages backwards, rotate the anterior angle to which the vocal cords are attached outwards, thus opening the glottis. This action, of course, is purely involuntary and is excited by and coincident with those impulses which set in play the other muscular contractions concerned in the inspiratory act.

According to the old Marshall Hall teaching this act was regarded as reflex, and due to the accumulation of carbonic acid in the pulmonary vessels. More recent experience, however, has shown it to be due to the accumulation of carbonic acid in the venous blood which circulates through the respiratory centres of the medulla. The respiratory function of the larynx therefore consists solely of this glottis-opening action, and is to an extent adventitious and rendered necessary by the location of the phonating apparatus in this cavity. While the inspiratory function of the larynx, therefore, has to do with the current of air in inspiration, the more important and intricate function of the larynx, viz., that of phonation, has to do with the ex-

piratory current; the two, however, are accomplished in a perfectly harmonious manner and without interference one with the other. In fact, they are to an extent supplementary to each other, the involuntary function of respiration taking place during the intervals of phonation, which is a purely voluntary function. This latter is accomplished simply by drawing the vocal cords together, thus narrowing the chink of the glottis and driving the current of air through this narrow chink in such a way that the cords are thrown into vibrations. These vibrations or phonating sounds are subsequently converted into articulate language by the movements of the fauces, tongue, and lips. The volume of sound is regulated by the force with which the air is driven through the narrow chink. The varying pitch which may be given to the voice is regulated by the tension of the vocal cords. One other characteristic which the voice possesses is that of timbre or quality. This is dependent upon the general contour of the upper air tract. When the vocal cords are thrown into vibration and sound is produced, of course the air in the whole of the upper air tract including the nasal chambers is thrown into vibration and a certain resonance is thus added to the voice and a certain quality. If, for instance, as the vocal waves emerge from the larynx they escape into the outer world through the mouth alone, we recognize a certain oral quality or absence of nasal resonance. If the tones are thrown into the nose we get a marked nasal resonance in the voice. A nice adjustment between these two is the characteristic of a pleasant and agreeably sounding voice. This, of course, is regulated largely by the position of the palate in phonation; if this is drawn well up against the pharynx the vocal tones emerge through the mouth and there is an absence of the nasal resonance, while on the contrary, if the palate is drawn down on the base of the tongue, the sound waves escape into the nose, thus intensifying this quality and giving a disagreeable twang to the voice.

The ordinary conversational use of the larynx does not call into play the highest and best powers of this organ, therefore it is often apparently unimpaired even when there is a considerable departure from a healthy tone in the laryngeal mucous membrane. The singing voice, on the other hand, brings into action the very best powers of the larynx and any deviation from the condition of health results in a noticeable impairment of tone. Furthermore, as we shall see later, this applies not only to the larynx itself but to the whole of the upper air tract. The recognition of the very close and intimate relation which exists between the different portions of the breathing apparatus is of the very greatest importance and essential to an intelligent comprehension of the disease processes which are met

with here. We are far too apt to seek in the larynx for the source of vocal impairment especially in the singing voice, and yet in by far the largest proportion of cases which come under our observation the primary source of trouble is to be found in some portion of the nasal or pharyngeal cavities. In other words, I am disposed to think that we are apt to overestimate the importance of the larynx as the primary seat of disease. Its mechanism is simple and its functions are easily performed, and, moreover, it maintains itself in a fairly healthy condition as long as the other portions of the air tract remain healthy. It becomes the seat of diseased action in the effort to surmount difficulties which are imposed upon it by imperfect functionating conditions in the regions above, or, perhaps still more frequently, catarrhal conditions above result in impairment of function and develop similar conditions in the larynx, which thus becomes the seat of disease secondarily rather than primarily.

LARYNGOSCOPY.

Laryngology, the term used to designate the comparatively new specialty which confines itself to the treatment of the upper air tract, dates its beginning from the discovery of a very simple process which enables us to bring under ocular inspection the whole of the upper air tract. It owes its origin to the desire on the part of physiologists to determine the direct source of voice production. Various efforts had been made, dating back to the early part of the eighteenth century, to inspect the larynx during life. The method which was finally successful was devised by Manuel Garcia, an eminent singing-master of London, who conceived the idea of placing an ordinary dental mirror in the back parts of the fauces in such a position that sunlight could be reflected down into the larynx, while at the same time an ordinary toilet mirror was held before the face in such a manner that the image cast upon this dental mirror would be seen by the eye. Garcia's experiment was entirely successful, and he thus determined by his observations that the true vocal cords were the source of voice production. He presented the results of his investigations before the Royal Society of London, in 1855, in a paper entitled "Physiological Observations on the Human Voice." To Türck, of Vienna, on reading Garcia's paper, came the suggestion that this simple procedure might be of great value in the diagnosis and treatment of disease processes in the larynx. He failed, however, to improve on Garcia's method of examination and hence failed to render the procedure of practical value. Czermak, of Pest, substituting artificial for natural light and availing himself of the recently discovered methods sug-

gested by Reute and Helmholz, of examining the interior of the eye by a concave reflecting mirror, constructed an apparatus which rendered the process exceedingly simple and fully available as a therapeutic and diagnostic resource. The process having been perfected, its use for a number of years was largely confined to the examination of the larynx, and hence the terms "laryngology" and "laryngoscopy" came into use, with the result that the larynx for many years occupied somewhat undue attention, although the same device enables us to examine with equal success what we now regard as a far more important part, viz., the upper air tract and nasal passages. The terms laryngology and laryngoscopy we now accept in their broadest sense as embracing the study and examination of the whole of the upper air passages.

The essential requirements for the practice of laryngoscopy are, first, a throat mirror; second, a good source of illumination; and third, a concave reflecting mirror. The throat mirror consists of a small, round mirror encased in German silver and attached to a slender wire stem fitted with a handle, the whole being six to seven inches in length. They are made in sizes of from three-eighths to one and one-eighth inches in diameter, and number from 0 to 5, as shown

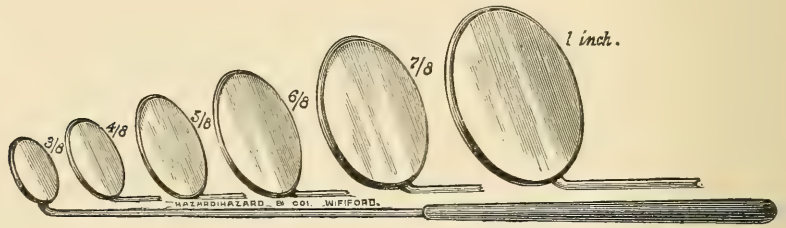


Fig. 39.—Throat Mirrors; actual sizes, from No. 0, three-eighths inch in diameter, to No. 5, one inch in diameter.

in Fig. 39. The mirror is attached to its stem for laryngoscopic purposes at an angle of 135°, and for rhinoscopic examinations at one of about 105°, the largest size being made for an examination of the larynx, while the smaller sizes are used for observing the nasal cavities and upper pharynx.

The light may be either the sunlight or artificial light, such as the lime light, gas, electricity, or a coal oil flame. In using gas, the Argand burner should be preferred as giving the steadier flame. The calcium light, of course, is a powerful illuminator though only attainable by a somewhat complicated apparatus. Where sunlight can be used it is by far the best illuminator attainable.

The reflecting mirror constitutes really the most important fixture of the laryngoscopic apparatus, and is practically the device which

has rendered laryngoscopy feasible. As we know, Reute conceived the idea of examining the fundus of the eye by reflecting rays of light from a plane mirrored surface. This process was subsequently greatly improved upon by Helmholz in the construction of the concave reflecting mirror by which the rays of light were converged. Furthermore, in order that the visual rays might coincide with the illuminating rays the concave mirror was perforated by a small aperture in its centre to enable the examiner to look along the central line of the illuminating rays, thus obtaining the most perfect observation at the point of the most perfect illumination. This reflecting mirror may be mounted on the head by an ordinary head band, such as Schrötter's, seen in Fig. 40, or the Pomeroy head band, as seen in Fig. 41. In each of these a small rounded knob projects from the back of the mirror near its border and fits into a ball-and-socket joint attached to the head

Fig. 40.—Reflecting Mirror, Mounted on Schrötter's Head Band.

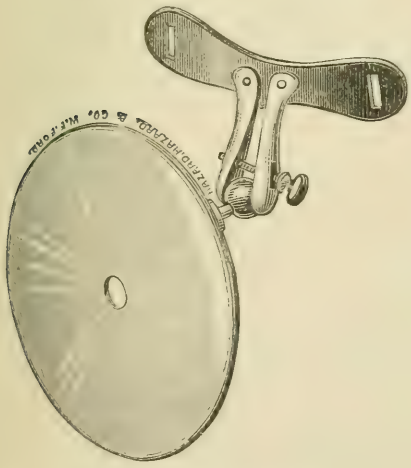


Fig. 41.—Reflecting Mirror Mounted on Pomeroy's Head Band.

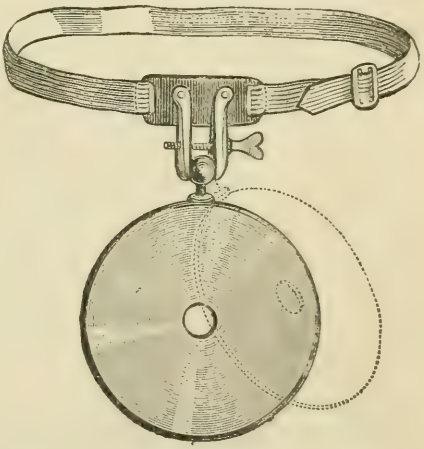


Fig. 42.—Bosworth's Head Band and Mirror.

band. A serious objection against this device lies in the fact that the lateral motion of the mirror is notably hampered. In Fig. 42 is shown a head mirror in which the ball is mounted on the periphery of the mirror, thus giving the widest freedom of motion—a very great

desideratum for facility in picking up light from whatever source it may be obtained.

In the early days of laryngoscopy these reflecting mirrors were made in sizes varying from four to six inches in diameter, thus they became heavy and somewhat cumbersome. My own preference is for the smaller mirrors varying from two and one-half to three and one-half inches in diameter, in that they are much lighter in weight, less irksome to the wearer, and practically give as thorough an illumination as can be wished, provided the source of light is a good one. The English method of mounting the head mirror on a spectacle frame, as shown in Fig. 43, does not present the simplicity of the head band. For office work I think the head mirror is quite as valuable as the fixed apparatus, although this latter device is preferred by many. This consists in mounting the reflecting mirror on

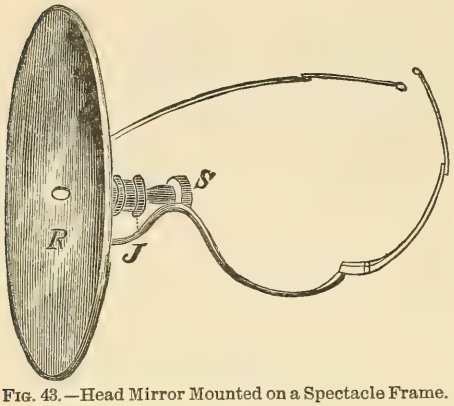


Fig. 43.—Head Mirror Mounted on a Spectacle Frame.

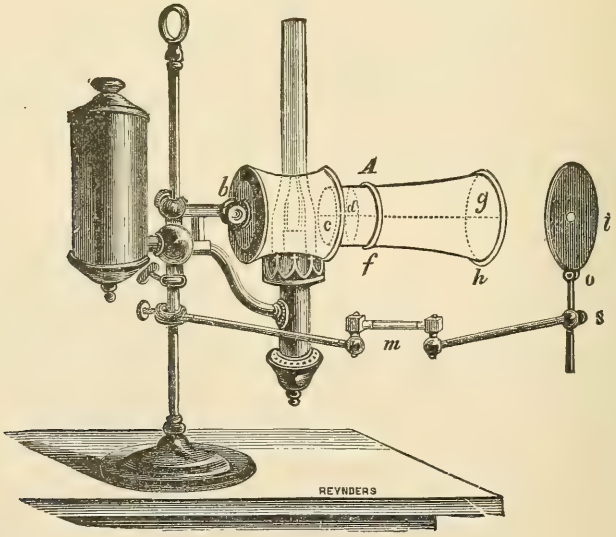


Fig. 44.—Tobold's Laryngoscope.

a flexible bar, which enables the operator to fix it at any point in front of the so-called laryngoscope, which consists of a metallic hood placed

over the artificial light to which it is attached. There is a perforation on one side of this hood to which is affixed either a single lens or a series of lenses. In Fig. 44 is seen the well-known Tobold laryngoscope attached to a German student lamp. The arrangement of the lenses is easily seen by the inspection of the cut. It is a convenient apparatus for office work, although there are no novel optical principles involved in its construction. The elaborate apparatus with which many specialists' offices are fitted up consist simply in an extension of the Tobold idea, as in the Sass laryngoscope and others. In Fig. 45 is shown the Mackenzie light condenser, mounted upon a movable gas fixture. Its method of use is easily appreciated by an inspection of the figure. It

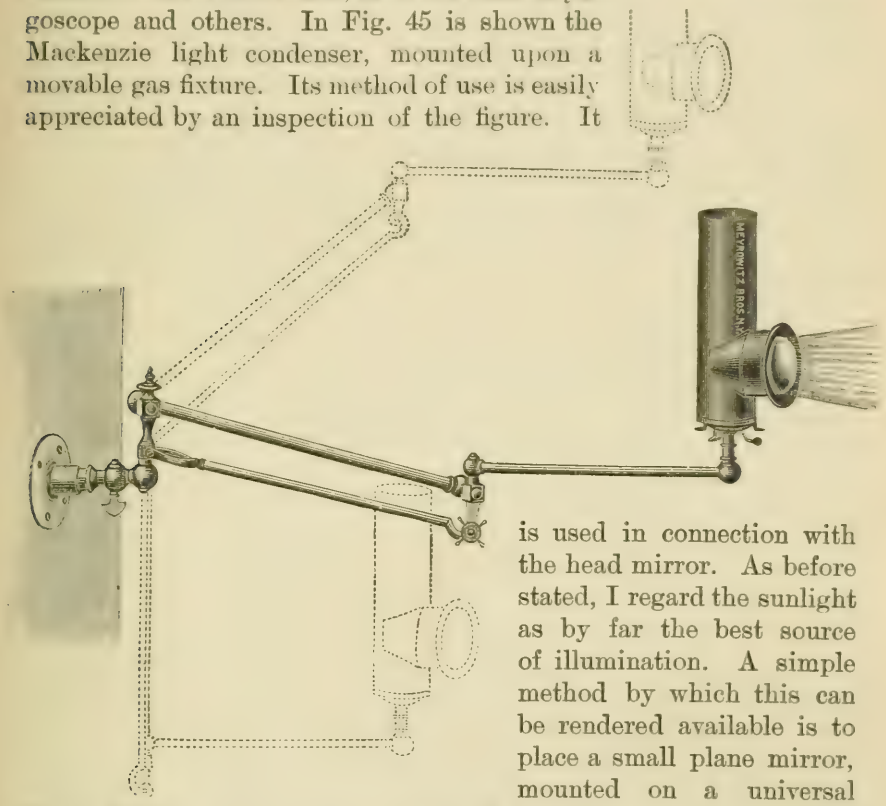


Fig. 45.—Mackenzie's Light Condenser, Mounted on a Ratchet Movement Gas Fixture.

is used in connection with the head mirror. As before stated, I regard the sunlight as by far the best source of illumination. A simple method by which this can be rendered available is to place a small plane mirror, mounted on a universal joint, in such a position that it may intercept the sun's rays and deflect them so that they fall on the reflecting mirror mounted on the head band.

In making a laryngoscopic examination, the patient is placed directly in front of the operator with the source of light over his right shoulder. The head mirror, if used, is then placed upon the head in such a manner that the rays of light will fall upon it, while at the same time the mouth of the patient can be easily observed by the right eye

looking through the perforation in the centre of the mirror; the mirror then, without disturbing this relation, is turned in such a way that a disk of light is thrown directly upon the mouth of the patient. This manipulation is not always easy to acquire and is only attained with a certain amount of practice, for in all cases the observer should not be hampered by an attempt to adjust the light by movement of the head. The light adjustment should always be made by movement of the mirror. Of course, in using the fixed apparatus this manipulation becomes simpler, but one should always acquire facility in the management of the head mirror, for its importance cannot be over-estimated, especially in making visits outside of the office.

In the selection of methods of examination I have no suggestions to make. There is no great advantage in any, certainly no superior advantage in the very elaborate apparatus which has crept into use in recent years. Knowing what one wishes to accomplish, the procedure becomes simpler as the apparatus is the less complicated. Of vastly more importance is the well-trained eye, which is acquired only by practice, and which enables one to recognize abnormal conditions with infinitely greater facility and certainty than any amount of perfected devices. Specially devised operating chairs were formerly a familiar feature among the laryngoscopist's office fixtures, but they possess no special advantage over the ordinary armchair with the back sufficiently high to afford the head of the patient a convenient rest.

In making the laryngoscopic examination, the patient being in the position already described, the mirror is warmed over the light sufficiently to prevent the moisture of the breath from being deposited upon it and thus obscuring the vision, and before introduction is touched to the hand or cheek, thus avoiding its use while too hot. The tongue of the patient is then protruded and seized between the thumb and forefinger, a napkin being inserted, and the tongue is drawn well forward, thus opening up the fauces, and to a certain extent dragging forward the epiglottis. The mirror (a No. 4 or 5 being used preferably for laryngoscopic purposes) is then taken in the right hand and held much as one holds a pen. Finally, it is introduced into the mouth, its handle being inclined downwards and outwards, its base being parallel to the dorsum of the tongue; it is then passed backwards without altering this relation until the edge of the mirror nearly touches the soft palate, the shaft of the mirror in this movement striking the angle of the mouth as a resting place and fulcrum. The subsequent movement consists in turning the mirror by twisting its shaft between the fingers until it is inclined at an angle of 45° to the line of vision, then it is carried backwards and downwards until the

uvula rests upon its posterior surface, when it is lifted boldly upwards and backwards until its lower edge comes entirely into view again and rests firmly against the posterior wall of the pharynx. The patient should then be directed to sound in a somewhat high key "*a*," which lifts the larynx and at the same time the epiglottis and exposes and brings into view the laryngeal cavity. The manipulation can be made easily and with facility without any special risk of gagging or irritating the patient, provided the movements are made deftly and at the same time firmly. An unsteady hand, of course, will always excite gagging or retching. Furthermore, the mirror itself should be kept in the median line, with its plane always at right angles with the field of vision. This position is well illustrated in Fig. 46. It is a matter of considerable importance that the mirror, when it is finally in position, should rest upon the posterior wall of the pharynx with its lower edge at a point above the free edge of the palate—in other words, that the soft palate and uvula should be crowded up firmly and vigorously as far as possible. This is of especial importance as the sensitive region of the pharynx—viz., that portion which if impinged upon will cause retching—is below the free border of the palate, impingement upon the portion above this point being less liable to excite reflex movement.

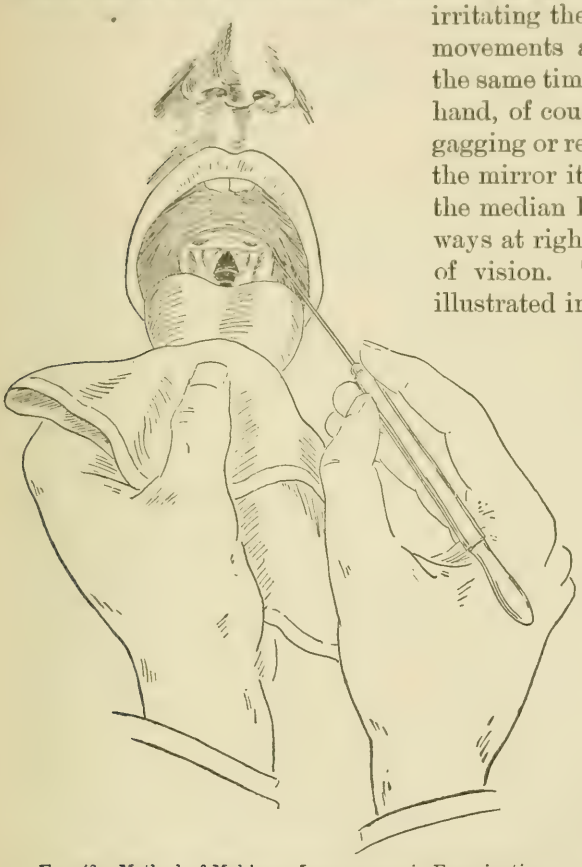


FIG. 46.—Method of Making a Laryngoscopic Examination.

The position of the mirror and the direction which the rays of light traverse are illustrated in Fig. 47. Commencing at the source of the illuminating rays as we see, their course is first to the reflecting mirror, then to the throat mirror in the fauces, then into the larynx, the return being from thence to the throat mirror and to the

reflecting mirror, reaching the eye through the perforation in its centre. We also have illustrated the importance of making use of this perforation, as in this manner the illuminating and the visual rays fall in the same line; whereas, if the observer places the reflecting mirror in front of the forehead looking beneath it, as is often done, the visual rays are liable to be reflected by the throat mirror on a portion of the larynx not fully illuminated.

In our older works on the laryngoscope are found detailed a long series of obstacles to laryngoscopy and the methods of overcoming

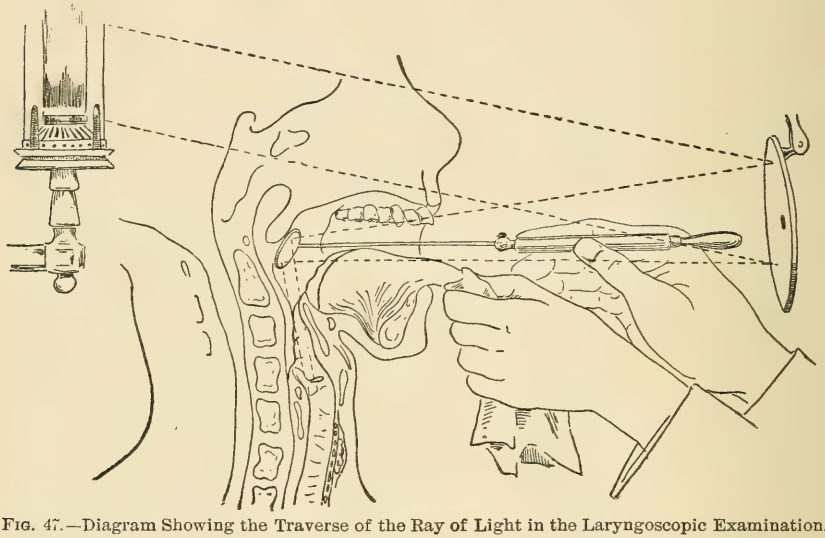


FIG. 47.—Diagram Showing the Traverse of the Ray of Light in the Laryngoscopic Examination.

them. I know of no practical value in these suggestions, in that it is rare that we meet with a patient who does not submit to a laryngeal examination, provided our manipulations are made with a steady and firm hand. This can be acquired only by practice. Of course, if the fauces are exceedingly sensitive cocaine may be resorted to, but this is not advisable on account of the great discomfort which is caused thereby in the pharynx.

We are usually taught in making an examination that the laryngeal image is reversed in the mirror. This reversal is simply the same as takes place when one looks into an ordinary dressing-mirror and need not be taken into consideration. Anatomy teaches the relation of the parts, and hence I think it is advisable for those who are commencing the practice of laryngoscopy to make themselves thoroughly familiar with the regional anatomy of the parts. This should be done preferably by means of the human larynx taken directly from the cadaver. It should be studied in connection with text-books

not only by direct ocular inspection, but also with the throat mirror in position.

In the human subject, the mirror being placed in position, and, as before stated, the sound of "*a*" being given to elevate the larynx and open its cavity, the first object that is noticed will be the epiglottis standing out prominently in the upper portion of the mirror, with its sharp crescentic edge and yellow fibrocartilage showing through the thin mucous membrane. On the anterior or lingual face of the epiglottis are seen three folds of mucous membrane stretching from it to the base of the tongue, one in the centre and one on either side. These are the glosso-epiglottic ligaments which divide the depression between the epiglottis and the base of the tongue into two fossæ, the lingual or glosso-epiglottic fossæ. The posterior face of the epiglottis is marked at about its center by a rounded pad-like prominence of a deep-red color which is called the cushion of the epiglottis. This possesses no special physiological or pathological interest. Passing downwards and backwards from either side of the epiglottis are two folds of mucous membrane which extend to the arytenoid cartilages, these latter being observed in the lower part of the mirror as two small, rounded knob-like prominences, which in the living subject are seen to move from a state of close proximity to one of wide separation in the acts of respiration and phonation. These folds of mucous membrane form the lateral walls of the larynx and are called the aryepiglottic folds. On either side of these folds are noticed the pyriform sinuses. In the posterior portion of these folds, immediately in front of the arytenoid cartilages, are observed two small projections, one on either side, made by the upper end of the cartilage or staff of Wrisberg. Surmounting each arytenoid cartilage and giving it additional prominence are the cartilages of Santorini, which, however, are not easily recognized in the laryngeal mirror.

Passing between the arytenoid cartilages and forming the posterior wall of the larynx is the arytenoid commissure. The circuit of the laryngeal cavity is thus completed by the epiglottis in front, aryepiglottic folds on either side, and the arytenoid cartilages and commissure posteriorly. Immediately behind the commissure we notice a closed fissure between it and the posterior wall of the pharynx which marks the orifice of the gullet. Immediately below the aryepiglottic folds on either side are seen the two ventricular bands. These are two folds of mucous membrane, one on either side, extending from the receding angle of the thyroid cartilage in front to the arytenoid cartilages behind. They are rounded, prominent folds of mucous membrane which present a somewhat deeper color than the other portions of the laryngeal cavity. They move with the aryte-

noids and are parallel with the vocal cords which are seen immediately beneath them, two white glistening bands of fibrous tissue covered by a mucous membrane so thin as to be practically transparent. They also extend from the receding angle of the thyroid to the arytenoid cartilages and project notably within the border of the ventricular bands. The entrance to the ventricles of the larynx lies between the ventricular bands and the true cords, though only recognized as a shaded line at this point. When the cords are separated in the act of respiration a small, knob-like projection is seen immediately in front of each arytenoid cartilage. This is the vocal process, and is formed by a cartilaginous prolongation of the anterior angle of the base of the arytenoid cartilage. When the cords are brought together, the space or opening between them forms the rima glottidis, or chink of the glottis. During the act of phonation when the cords are in apposition this chink is merely a straight line extending from the receding angle of the thyroid cartilage to the arytenoid commissure. During the act of phonation the cords are brought

FIG. 48.—The Laryngeal Image during Phonation.

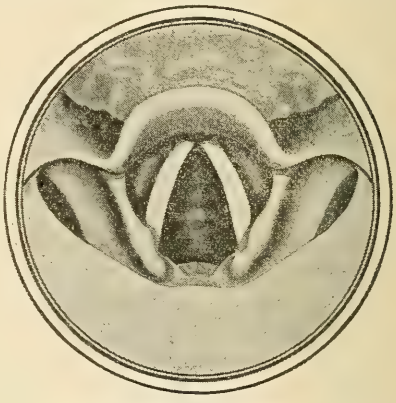


FIG. 49.—The Laryngeal Image during Inspiration.

into close approximation, as seen in Fig. 48, and thus a view of the parts below is prevented. During the act of inspiration, however, the cords are widely separated posteriorly and the subglottic portion of the larynx and the rings of the trachea may be brought under inspection (see Fig. 49), and in a favorable case even the bifurcation.

In a general way the appearance of a normal larynx differs in no especial manner from the healthy mucous membrane seen in other portions of the body. It is of a light rose-pinkish color with a tendency to a yellowish tinge especially where the cartilages are seen through the transparent membrane, as at the crest and sides of the epiglottis and at the prominence made by the cartilages of Wrisberg

and Santorini. On the other hand, where the membrane covers glandular or areolar tissue, it is of a deep-red color, as that of the cushion of the epiglottis, the aryepiglottic folds, the ventricular bands, and the arytenoid commissure.

In making a laryngoscopic examination we first study the general contour of the parts to notice any deviation, then inspect the mucous membrane and observe any departure from the normal pinkish tinge of health, and finally the movements of the cords may be observed in phonation and respiration to determine their perfect approximation and their readiness of abduction, and furthermore, whether their movements are symmetrical. In making this latter observation it should always be borne in mind that not uncommonly the epiglottis is deflected to one side or the other, and that in attempting to adjust a line between the centre of the epiglottic crest and the chink of the glottis, the whole laryngeal image may be distorted. Hence our mirror should be placed in such a position as to give a symmetrical image of the lower portion of the larynx without reference to the position of the epiglottis, for in this manner only can we determine accurately as to the symmetrical movements of the cords.

In discussing diseased conditions as we meet with them in the larynx we shall have to do with, (1) Inflammations; (2) local manifestations of constitutional diseases; (3) neuroses; (4) accidents and injuries; and (5) benign and malignant tumors.

INFLAMMATIONS OF THE LARYNX.

In this section the nomenclature adopted by the writer in his previous publications will be followed. The essential feature of this consists in making the name of the disease express as far as possible its character and location. The termination "itis" in all cases refers to catarrhal, in contradistinction to croupous and diphtheritic inflammation, the latter form being designated when referred to.

Acute Laryngitis.

In the older literature this term was used to designate an acute inflammatory process in the laryngeal mucous membrane which assumed a phlegmonous or œdematous type, and the name subacute laryngitis was used to designate a mild inflammatory process of a catarrhal character in which no tendency either to œdema or phlegmon was manifest. We reject the term subacute as being somewhat indefinite, and prefer to describe under the designation of acute laryngitis that form of inflammatory action which, while mild in

character, still constitutes an acute process. The affection is comparatively a trivial one, its principal interest lying in the fact that the voice is liable to be rendered either hoarse or completely aphonic, a symptom to which the laity usually attach unnecessary importance.

Etiology.

Most acute inflammatory processes involving the air tract are generally regarded as primarily the result of a cold. Furthermore, we are usually taught that as the result of repeated attacks of inflammatory action a chronic inflammatory process sets in. My own experience teaches me that the true sequence of events as regards the upper air tract is just the reverse of this, and that in the large majority of instances, if not in all cases, a cold, so called, resulting in a mild inflammatory action of the larynx or of any other portion of the upper air tract, is merely the lighting up of a chronic process in consequence of an exposure, the chronic inflammatory process having developed slowly and somewhat insidiously as the result of diseased action above, viz., in that most important and intricate portion of the upper air tract, the nasal chambers. In addition to this I am disposed to think that an inflammatory process in the larynx is in the large majority if not in all instances secondary to some disturbance in the nasal cavities. Every breath of air taken into the lungs is designed by Nature to pass through the nasal cavities, where it is warmed and cleansed, and what is of still greater importance, becomes thoroughly saturated with moisture by passing over the turbinated bodies, constantly coated as they are in health by the watery serum which escapes from the venous sinuses by the process of exosmosis. Given, then, some obstructive lesion in the nose or a chronic inflammatory process which interferes with this important function and the result is a current of air reaching the delicate structures of the larynx and air passages improperly cleansed and insufficiently moistened; the necessary result is irritation of the parts and eventually the establishment of a mild chronic inflammatory condition ready to be lighted up into an acute inflammation on any slight exposure to cold.

In addition to lesions of the nasal cavities, we can easily understand how a morbid condition of the naso-pharynx, such as the existence of an adenoid growth or of a naso-pharyngeal catarrh, may also result in the development of a morbid condition in the larynx. The important feature of all this is the recognition of the fact that in any given case of acute laryngitis we should understand the development to be really the result of a chronic process which also demands treatment, and furthermore, of the existence of some departure from the normal state in the parts above. As a matter of clinical observa-

tion the result of a cold in early life is usually an attack of acute coryza. As this occurs with more or less frequency we find in connection with a cold in the head or immediately following it, there occurs an attack of hoarseness. As the years go on the tendency to colds in the head disappears to a certain extent, and as the result of an exposure the patient suffers from attacks of acute naso-pharyngitis or laryngitis or perhaps bronchitis, the primary seat of the chronic process having been in the nose while the involvement of the air passages below has become its direct result. Acute laryngitis may occur at any period of life. Before puberty it is probably in most instances secondary to some disease process in the lymphatic structures, such as hypertrophy of the faucial or pharyngeal tonsils. In early manhood it is probably secondary to some form of nasal stenosis, such as deflection of the septum or hypertrophic rhinitis. In the later years of life it is met with in connection with chronic naso-pharyngeal catarrh. In singers in adult life it is not infrequently the accompaniment of an enlargement of the lingual tonsil. It may also be the result of the inhalation of irritating vapors, or perhaps a dust-laden atmosphere, and may be brought on by overtaxing the voice in speaking or singing. It also occasionally follows an attack of measles, scarlet fever, or other of the exanthemata. The administration of iodide of potassium produces often a hyperæmia of the larynx which is scarcely, however, to be regarded as an inflammatory process.

Pathology.

Like all inflammatory processes the first stage of the attack is characterized by a dilatation of the blood-vessels and the arrest of secretion, vascular plethora being more marked in those portions of the mucous membrane which are the least firmly attached to the parts beneath. After the first twelve or twenty-four hours there is a moderate amount of secretion, consisting largely perhaps of a serous exudation, giving relief to the hyperæmia. The inflammatory process is mainly confined to that portion of the larynx which is above the vocal cords, these latter being slightly reddened but never to the extent of more than slight tumefaction, owing to the exceeding tenuity of the mucous membrane. The morbid process does not usually extend to the epiglottis.

Symptomatology.

Impairment of voice, of course, is the one prominent and most notable symptom which characterizes an attack of acute laryngitis. This may be very slight in amount, giving to the voice a slightly metallic ring, or if the mucous membrane covering the cords or below

the cords is much injected, the voice may be completely lost, although this latter is exceedingly rare in a simple process, confining itself mainly to the laryngeal cavity. Complete aphonia is more liable to occur where the whole of the upper air tract is involved. Cough is occasionally present. This phenomenon as we know is a reflex effort whose object is to expel a foreign body from the air passages. In a catarrhal inflammation the foreign body is an accumulation of mucus, and the secretion in this affection being but limited in amount, cough is not demanded for relief. When the subglottic larynx and the upper part of the trachea are involved, the cough may be at times very harassing and persistent. When the secretion is limited or tenacious in character the futile efforts at expulsion render the parts sore and add much to the discomfort of the patient, otherwise the discussion of the symptoms of an ordinary attack of acute laryngitis has to do mainly with the simple dysphonia.

The disease is usually a local one and any febrile disturbance which accompanies it is generally of a somewhat trivial character unless other portions of the air tract are involved.

Diagnosis.

The impairment of voice and the mild general symptoms which accompany the attack are usually sufficient to render a diagnosis comparatively easy, although in all cases, of course, it is well that a laryngoscopic examination should be made, if for no other purpose than thoroughly to exclude the possibility of some graver disease having attacked this delicate and sensitive region; for while of course in a very large majority of instances mere hoarseness or even loss of voice is a comparatively trivial symptom, we must bear in mind that some of the very grave and dangerous affections come on at times in a very insidious way. The laryngoscopic examination reveals the mucous membrane lining the whole larynx, presenting the bright-red appearance characteristic of an acute inflammatory process, this redness being especially marked in those parts where the mucous membrane is most loosely attached. The cords present merely a pinkish or a light reddish tinge, owing to their fibrous structure showing through the thin membranous mucous covering. The movements of the larynx are not involved, and approximation of the cords is perfectly accomplished, unless the arytenoid commissure is the seat of a notable tumefaction, thus preventing the approximation of the cartilages. If the inflammatory process is of a severe type we may have a certain degree of infiltration of the muscles which interferes with vocal tension and renders approximation somewhat sluggish of accomplishment; this, however, is rare. A uniform bilateral

injection of the mucous membrane, with the absence of any localized tumefaction, together with the other evidences of inflammatory action which are usually noticed in the other portions of the upper air tract, will usually be sufficient to render a diagnosis simple. In many of the graver affections of the larynx, such as syphilis, tuberculosis, paralysis, and tumors, impairment of the voice is the first symptom to which our attention is called. In syphilis the voice is hard, coarse, and somewhat raucous in character. In tuberculosis it is soft in tone, its prominent feature being a phonatory weakness, as it were. In most forms of paralysis the voice is simply weak and not hoarse, while in tumors it is usually cracked and broken. Furthermore, the vocal impairment is slowly progressive and does not come on suddenly.

Course and Prognosis.

An attack usually runs its course in from five to eight days and terminates spontaneously unless the organ is subjected to unusual strain and over-use while in a state of inflammation. It, of course, involves no danger to life, and the gravity of the situation lies in the temporary impairment of function which in the ordinary callings of life is not important. It only becomes serious to public speakers and singers, or to those whose calling is dependent upon the perfect use of the voice. There is no tendency to extension, as is popularly supposed, to the chest, and if such extension is noticed it is to be attributed rather to the severity of the inflammatory process in the nose and naso-pharynx which may accompany it.

Treatment.

The first indication for treatment is based on the recognition of the fact that the disease is usually secondary to trouble in the parts above, and hence our first efforts should be directed to controlling the disease process in the nose or naso-pharynx. If it supervenes upon an attack of acute rhinitis, these parts should be thoroughly anæsthetized and exsanguinated by a four-per-cent. solution of cocaine, after which I know of no better treatment than the application of a small crystal of chromic acid to the most prominent portion of one or the other turbinated bodies, thus pinning the membrane down as it were by an inflexible slough, which thus holds the turgescence in check and prevents a refilling of the venous sinuses. In addition to this I have no hesitation in prescribing for the patient's own use a very mild solution of cocaine in connection with some cleansing and alterative lotion, such as the following:

℞ Cocainæ muriatis, ... gr. v.
Zinci sulpho-carbolatis, ... gr. ij.
Sodii biboratis, ... gr. v.
Sodii bicarbonatis, ... gr. iij.
Aquæ, ... ʒ i.
M. This is to be applied to each nasal cavity every two hours.

I do not hesitate to say frankly in this connection that I regard the dangers of the local use of cocaine as greatly over-estimated. A one-per-cent. solution of cocaine as in the above, I think, may be safely used to tide over the ordinary cold without incurring the risk of the so-called "cocaine habit." Whether such exists or not I do not know. I certainly never met with one where it was anything more than an indulgence perhaps, rather than a serious or dangerous habit. It undoubtedly may produce wakefulness and pleasant stimulation and nervousness in much the same way as will strong tea or coffee. Where such is observed I certainly should interdict its use, otherwise after an experience of ten years with the drug I see no reason why we should not avail ourselves of its extraordinary and invaluable properties.

If the laryngeal invasion is accompanied by an involvement of the naso-pharynx, this cavity should be thoroughly douched by means of the post-nasal syringe, with a weak solution of soda or salt, to which has been added a little carbolic acid or listerine, after which it is well to apply directly to the parts either through the nose by means of a probe or cotton pledget, or through the mouth, a solution of nitrate of silver of the strength of from gr. x.–xv. to the ounce. This application may also be made to the oro-pharynx if this is involved. If the attack occurs in connection with hypertrophy of the faucial or pharyngeal tonsils these parts should be thoroughly cleansed as far as possible, and this cleansing be followed by an application of either compound tincture of iodine or glycerole of tannin. These measures, if they serve to control the diseased action in the parts to which they are directed, will in many cases be quite sufficient to control the inflammatory process in the larynx, although local applications to the latter will always serve to hasten the resolution of the attack. Curiously enough many prominent writers object to local applications to the larynx in acute laryngitis. Why this should be I fail to understand, unless it be to the old method of making these applications by means of the brush or probang. These instruments, I think, should never be used in the larynx except in the more aggravated forms of disease, such as persistent and long-standing chronic laryngitis, or lupus, or syphilis, or some of the graver affections, but never to the organ in a state of acute inflammation. When

this latter exists I think the only proper method of making applications should be by means of the atomizer worked by the ordinary rubber hand bulb, or, better, the compressed air apparatus. By this means we not only avoid the injury to the parts which might result from the rude impact of the probang or brush handle, but accomplish our purpose with the least possible irritation to these delicate structures. In this way we may use one of the following in the order of preference:

Liquor ferri persulphas,	.	gtt. v. to x. to the ounce.
Argenti nitras,	. . .	grs. ij. " v. " "
Zinci sulphas,	. . .	grs. v. " x. " "

Before using these, it is well, perhaps, to cleanse thoroughly the laryngeal mucous membrane by the application of a carbolized alkaline solution such as Dobell's, or even a simple solution of borax or salt. The steam atomizer in former years has had a somewhat extensive vogue in the treatment of laryngeal disorders, but while it is a very attractive piece of mechanism I have always doubted whether it accomplished any favorable result in these cases, although it may give a certain amount of relief to certain subjective symptoms during the early part of the attack when the mucous membrane is dry and the parts are stiff and painful. There are a number of resinous gums and volatile oils such as compound tincture of benzoin, camphor, oil of turpentine, oil of peppermint, oil of tar, creosote, oleoresin of cubebs, and others, in which the active principle is rendered to a certain extent volatile by the action of hot water. The use of these frequently repeated often gives marked relief in the early stage of the attack. A teaspoonful of any one of these may be added to half a pint of hot water in an open-mouthed bottle, this being held to the mouth; the fumes arising from it can be carried well into the upper air passages by the ordinary respiratory act. If the discomfort is notable this procedure may be carried out by the patient at home at intervals of every two or three hours. There are two ways of relieving an acute inflammatory process in the mucous membrane. One is by the direct application of astringents such as already described, their action being to correct secretion without controlling the hyperæmia. Another method is by resort to stimulation, and this is probably the action of the resins and volatile oils last mentioned. What is accomplished by stimulation is to excite a somewhat freer secretion of mucus or sero-mucus. A condition which is oftentimes most distressing and uncomfortable to the patient is one where the membrane is dry and clogged with thick inspissated mucus which voluntary efforts fail to dislodge. It is in these cases that the

greatest relief is afforded by stimulation, and this is probably the way that relief comes from the last-mentioned drugs. Such external applications to the neck as poultices, hot fomentations, or counter-irritants probably do more harm than good in acute laryngitis, certainly they add much to the discomfort of the patient, and they are usually to be avoided. Cold applications in the form of wet compresses, if applied diligently and changed so frequently that they continue to be cold applications, are often attended with marked relief, and if commenced early enough in those cases in which the larynx is the seat of considerable inflammation may serve to abort the attack. The applications should be changed, however, every twenty to thirty seconds in order to be effective. In connection with cold compresses the patient may also take ice lozenges internally at the same time. The ordinary throat lozenge which is sold in the drug stores is composed of a small amount of some anodyne, stimulating, or expectorant drug, incorporated either in cane sugar or a fruit juice. These are taken in the mouth and allowed to melt, when the drug in the act of swallowing is supposed to come into contact with the inflamed mucous membrane. If the pharynx is involved this undoubtedly occurs; but certainly the drug does not come into contact with the mucous membrane of the larynx.

Rhatany, krameria, aconite, chlorate of potash, tannin, gallic acid, cubebs, lactucarium, muriate of ammonia, etc., are among the drugs used in this manner. The astringents mentioned here are, I think, somewhat objectionable and inefficient. Muriate of ammonia, cubebs, peppermint, and others of the mint series, are stimulants. Their effect is to excite serous exosmosis, hence their administration whether in the form of a lozenge or otherwise is one from which excellent results may be expected. Their action is not directly local, but follows their absorption into the blood and subsequent excretion through the mucous membrane of the fauces. The internal administration of medicine is not ordinarily called for, although it is well, at the onset of the attack, to administer a mild saline laxative and, as in most cases of ordinary cold, to give two grains of quinine, or, better still, ten grains of salicin three times daily. If the larynx is the seat of much irritation some mild sedative such as lactucarium may be given. An excellent form for the administration of this is in the throat lozenges sold in the drug stores under the name of Pâte Aubergier. If this fails to relieve, codeine may safely be administered in doses of from gr. $\frac{1}{6}$–$\frac{1}{4}$ every four hours.

We are often called upon by public speakers or singers seeking relief from a cold or hoarseness, the result of an attack of acute laryngitis, and our professional skill is not infrequently severely tested in

our effort to restore their vocal cords to a proper condition of function in a limited time. There are certain measures, however which can be enforced with excellent promise of good results. These in the order of importance may be stated as follows: 1st, Perfect and absolute rest to the voice, the patient being enjoined to confine himself absolutely to a whisper and even to avoid this as far as possible; 2d, Confinement to one's room, which should be kept at an equable temperature of from 68° to 70° F. The room should be properly ventilated, though, of course, with the avoidance of draughts; 3d, The atmosphere of the room should be kept thoroughly charged with moisture by means of a cup of boiling water over a spirit lamp; 4th, The external application of cold should be made by compresses to the neck, or perhaps the use of Leiter's coil; 5th, A five-grain solution of nitrate of silver should be sprayed into the larynx twice or perhaps three times in the course of twenty-four hours; 6th, If an acute rhinitis exists, this should be reduced and kept under control by the application of chromic acid to the turbinated bodies after the manner already described; 7th, If the attack is accompanied by an acute inflammation of the tissues of the naso-pharynx, this cavity should be thoroughly douched with a solution of borax or soda or salt in warm water, applied by means of the post-nasal syringe, after which a twenty-grain solution of nitrate of silver should be applied to the parts once daily. Furthermore, for its specific influence on the inflammatory process in the naso-pharynx, the patient should be put under the influence of aconitine, gr. $\frac{1}{50}$ being given every hour until three doses have been taken, and afterwards every three hours for the remainder of the day, or until its constitutional effects have been experienced, such as prickling in the fauces or tingling of the lips; 8th, A half tumbler of Hunyadi or Villacabras water should be administered, and a free action of the bowels obtained, this being specially indicated if the naso-pharynx is involved. This may seem a somewhat elaborate and rigid course of treatment and is one which patients will not ordinarily submit to for an affection which involves no greater danger or discomfort, yet in those cases where the use of the voice at a certain time is a matter of importance the patient will usually not hesitate to subject himself to it, and in many cases the result will be restoration of function to the larynx at the end of from twenty-four to thirty-six hours, whereas otherwise the attack might have lasted several days.

Acute Laryngitis in Children.

An acute inflammatory affection involving the mucous membrane of the larynx in children requires a separate consideration from that in adults, owing to the fact that the organ is smaller in its calibre and the mucous membrane which lines it is more vascular and rather loosely attached to the parts beneath; hence the morbid process in this region is liable to assume a somewhat graver aspect and pursues a somewhat different course. Moreover, the development of the disease assumes a different character as the inflammatory process involves the membrane above the glottis or the membrane below, giving rise in the one case to what we may designate as an acute supraglottic laryngitis and in the latter case an acute subglottic laryngitis; the former constitutes a disease which runs a somewhat mild course and involves no great danger to life, while the latter is liable to develop alarming symptoms, not only on account of the laryngeal stenosis but also owing to the fact that it occurs in connection with certain systemic symptoms. Acute laryngitis in childhood may occur at any time under the age of fourteen, although it is more liable to occur between the ages of one and four.

Acute Supraglottic Laryngitis.

This forms a disease different in no sense or degree from that which occurs in adult life and consists in a mild form of inflammatory action involving that portion of the mucous membrane covering the vocal cords. Any mild catarrhal inflammation of the air passages above may act as a predisposing cause, although, perhaps, its most active predisposing cause is to be found in those morbid processes in the lymphatic structures which result in hypertrophy of the faucial or pharyngeal tonsils. Its exciting cause, of course, is usually some simple exposure to cold. Its development is usually marked by a mild, febrile disturbance with loss of appetite, or it may develop with apparently no systemic symptoms, the inflammation being confined mainly to the mucous membrane covering the vocal cords. The voice is somewhat hoarse or the child may become completely aphonic. Cough is not present in this form of the disease as a rule, although the child may complain of a sense of tickling or irritation in the throat. The mucous membrane of the larynx is not much thickened and the movements of the palate are not interfered with, although closure of the glottis may be impaired if the membrane covering the arytenoid commissure is notably injected, as may be the case. Dyspnœa or spasm of the glottis is not met with, this symptom belonging more par-

ticularly to the subglottic form of the disease. We find, therefore, that the affection is of a somewhat trivial character, but owing to the gravity which attaches in the mind of the laity to hoarseness in children and the apprehension which is always excited by it, the important question which arises in connection with such an attack becomes that of establishing a diagnosis. The mild form of the febrile disturbance or the absence of it, the character of the voice, which is hoarse or metallic rather than completely lost, and the absence of dyspnœa should ordinarily serve to allay apprehension, especially if an attack occurs in connection with an acute rhinitis or acute inflammation of the pharyngeal tonsil. It is of special importance to test the character of the voice, complete loss of which is always to be regarded as of greater importance than simple hoarseness. Moreover, tenderness on external pressure about the region of the larynx is to be regarded as rather symptomatic of the disease under consideration. If it is possible, of course, a laryngoscopic examination should be obtained of the parts, and this is by no means as difficult as is usually supposed. If the fauces do not tolerate the manipulation of them, as a rule a momentary glance at the larynx with the mirror in position can be obtained by exercising a little force and persistence, that is, by holding the tongue until the child in his struggles gags or coughs, when the epiglottis and the larynx are elevated, and a momentary view may then be obtained of sufficient length to show the absence of any serous infiltration or membranous deposit.

The disease runs its course in from five to ten days and undergoes resolution. The only danger which is to be considered is the possible occurrence, as a complication, of subglottic laryngitis. This is especially liable to occur in children of the lymphatic habit, as manifested by the existence of enlarged faucial or pharyngeal tonsils. It is to be borne in mind, of course, that the first stage of a croupous or diphtheritic inflammation is a simple catarrhal process, and while at the onset of a hoarseness in a child it is impossible to decide that this may not be threatened, yet, as we know, the commencement of the graver process is usually marked by far more severe constitutional disturbance in the way of febrile movement than is that of the simple process. Furthermore, the membranous deposit makes its appearance in a comparatively short time, usually in a few hours after the commencement of an attack. While we regard the disease as of a somewhat trivial character, I think we should always bear in mind that a mucous membrane in a state of acute inflammation is far more liable to afford a lodgment for a disease germ than is one in a state of health, hence a child with an acute laryngitis of a mild form is to a certain degree rendered more susceptible to croup or diphtheria than one in

health. Although I do not consider this danger a grave one clinically, yet I think it is one to be kept in mind. This also should lend weight to the view that it is not well to neglect even a mild attack of hoarseness in a child. If it occurs in connection with acute rhinitis this should be brought under control by the use of the following:

℞ Cocainæ muriatis, ... gr. ij. to iv.
Aquæ, ... ♏ xv.
M. fiat solutio et adde:
Glyceriti acidi tannici, ... ʒ i.
Olei petrolati, ... ad ℥ i.

This should be applied to the nasal cavities by means of an atomizer every two or three hours. If in connection with it the pharyngeal tonsils are in a state of acute inflammation and the nasal cavities in consequence are filled with muco-purulent secretion, this should be removed by means of an atomizer, using a mild solution of salt or borax. Direct applications to the larynx in children are not easily made and as a rule scarcely necessary in this affection. The inflammatory process is sufficiently controlled by the internal administration of small doses of muriate of ammonia, cubebs, or some one of the mint series, after the manner already described in the section on acute laryngitis in adults. External applications to the neck and over part of the chest, such as camphorated oil or oil of amber, will be found particularly efficacious. Steam applications are not indicated and accomplish no good, although it is well that the child should be confined to the nursery which is to be kept at an equable temperature with the air thoroughly moistened by boiling water over a spirit lamp.

Acute Subglottic Laryngitis.

In the earlier books we have three forms of croup described—membranous croup, which is characterized by false membrane, which under our own more definite nomenclature we now call croupous laryngitis; spasmodic croup, which we now more properly designate spasm of the larynx; and catarrhal croup, which I take to refer to any form of acute laryngitis in children. Under the more definite nomenclature which we adopt, wherein the name of the disease defines both its character and location, we reject the old names and under the term of acute subglottic laryngitis in children designate that form of acute catarrhal inflammation of the larynx in which, while the supraglottic portion may be but slightly involved, the brunt of the inflammation is borne by that portion of the membrane which lies below the vocal cords, giving rise to swelling and to infiltration of the parts

to such an extent as to cause notable, and oftentimes alarming, dyspnœa. This is the disease, moreover, I think, which has been described in literature under the designation of false croup, spasmodic laryngitis, laryngismus stridulus, and by writers of the last century as Millar's asthma and thymic asthma.

Etiology.

The attack is brought on as in other forms of acute inflammation by exposure to cold, but I think when we seek still further for an explanation, why in the one case an exposure to cold results in a simple laryngitis, and in another in this more grave form of subglottic involvement, we shall find that in the latter case there is some constitutional tendency or habit which acts as a predisposing cause. We are told that it occurs with equal frequency among strong and healthy children and those which we speak of as delicate or weakly. This may be apparently true, yet I think if we look closely into the clinical history of these cases we shall find that those children who are called croupy—that is, those children who are liable to frequent attacks of croup—show evidences of what Potain called lymphatism, viz., a tendency to involvement of the lymphatic tissue in morbid action, whether these tissues be the lymphatic glands of the neck or the lymphatic bodies which go to make up the faucial or pharyngeal tonsil. Potain, I think, very cleverly described this condition of lymphatism as "not constituting scrofula but as constituting a condition which if carried one step farther would result in the true strumous condition." Now, as we know, the lymphatic structures in young children are especially liable to undergo hypertrophic changes, moreover, this liability is notable where these structures are aggregated together in masses as in the faucial and pharyngeal tonsils. Sappey has very clearly demonstrated that the lymphatic structures in the upper air tract of children are far more numerous than in adult life. One of the regions which is characterized by notable lymphatic aggregations is that portion of the larynx which is immediately below the vocal cords. The mere presence of these lymphatic structures in a state of chronic inflammation or undergoing hypertrophic changes is sufficient to excite a mild chronic inflammation of the mucous membrane covering them. Now, we have already asserted that as a rule a chronic inflammation is the first to occur in the mucous membrane and that repeated attacks of acute inflammation become later the prominent symptom. I believe that this is what occurs in the disease under consideration, and that in the so-called croupy children the mild chronic affection is present and to a certain extent latent until the acute process is brought on by some exposure; while,

therefore, I regard the lymphatic habit as by far the most active of the predisposing causes of the disease, it may undoubtedly arise in cases where this is not present, as the result of improper nourishment, bad hygienic surroundings, insufficient clothing, and general causes of this character. Gerhardt regards enlarged tonsils as a frequent cause of the disease. I think we reach a better appreciation of the disease by considering the enlarged tonsils and the subglottic laryngitis when present as both manifestations of the same general cause, viz., lymphatism. It is rather more common in boys than in girls. According to Guersant's experience it is more frequent between the ages of one and eight, while Rilliet and Barthez have not seen it before the age of three; Dehio has met with it up to eleven years of age.

Pathology.

The changes which occur are those which are met with in the ordinary cases of acute laryngitis with this addition, that we have a notable activity of the croupy process occurring in that portion below the cords where necessarily any encroachment, by their turgescence, on the lumen of the air passages involves more or less stenosis with the result of producing dyspnœa. This cannot be accounted for by the ordinary swelling which occurs as the result of a simple catarrhal inflammation unaccompanied by œdema; now œdema is not met with in this region, hence this stenosis is explained by the tumefaction or hypertropic or inflammatory changes which occur in the lymphatic structures which, as I have already stated, are so liberally distributed in that portion of the larynx which lies below the vocal cords.

Symptomatology.

The attack usually comes on as the result of an acute inflammatory process involving the mucous membrane of the cavity of the naso-pharynx; it may, however, be independent of the parts above. The general symptoms are anorexia, a general malaise, an elevation of temperature, perhaps to 100° or 101° F. The child is listless and shows a lack of interest. There is a change in the voice, not at first a hoarseness but rather a change in quality. There is a higher-pitched tone; later the voice becomes hoarse or there is complete aphonia. Cough is an early symptom in the attack, and it is recognized as croupous; it is harsh and barking in character. We have, to account for this, in place of the usual closure of the glottis, an artificial subglottic closure of the œdematous tissues, and while the child may be perfectly aphonic we yet have a high-pitched, barking, cough sound. Later in the disease there is some secretion, perhaps enough to cause some expectoration, but the cough retains the

croupy sound. With the development of the subglottic obstruction a crowing inspiration is noticed and the cough becomes more paroxysmal in character. Often there is an absence of symptoms through the day with pronounced exacerbation of the paroxysmal cough and dyspnœa at night. This may well be accounted for by the increased swelling of the tissues and by the accumulation of mucus which has become dried from mouth breathing. We do not need to suppose that true spasm of the glottis has occurred. The relief that is afforded by the dislodgment of the accumulated mucus bears out this view of its cause. Obstruction to the breathing to a greater or less degree may be noted during the whole course of the attack. The duration of the disease may be for one or two weeks. In children subject to it attacks are liable to recur, particularly during the spring and autumn months.

Diagnosis.

An effort should always be made to obtain a laryngoscopic examination. This can be done oftener than is generally supposed even in the case of very young children. We shall find the laryngeal mucous membrane reddened and swollen, and beneath the true vocal cords a bulging of the mucous membrane—a bellying-out, as it were—a striking and characteristic appearance (Fig. 50). The vocal cords and the larynx above may present an almost normal appearance or may be but slightly reddened. In the absence of laryngoscopic examination the diagnosis is rendered more difficult. The presence of diphtheria or croup must be eliminated. In distinction from diphtheria, we do not have the symptoms of sepsis. In diphtheria we look for the false membrane in the fauces. In membranous croup the dyspnœa is progressive, the fever is higher, the cough has not the same characteristics, the voice is lost early, and the cough is almost noiseless. Differential diagnosis may be exceedingly difficult and must be based upon objective rather than subjective signs. The nocturnal attacks in subglottic laryngitis are characteristic, while the slower but progressive course of membranous croup will serve to distinguish it. The involvement of the cervical glands may occur in all three diseases but is more prominent in diphtheria. The presence of a foreign

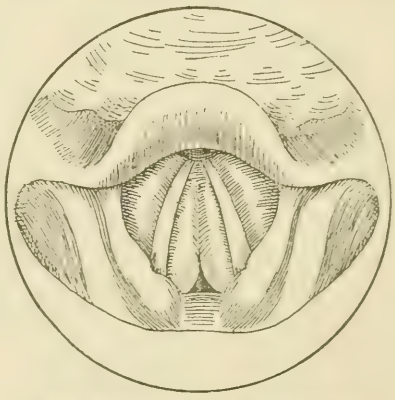


Fig. 50.—Acute Subglottic Laryngitis.

body may give rise to symptoms which resemble an attack of laryngitis. The laryngoscopic examination or the development of symptoms should soon make clear the diagnosis of this condition.

Prognosis.

The danger to life, in spite of the alarming character of the symptoms, is not serious. The rule is to have two or three nocturnal recurrences of dyspnœa, when they cease. The cough continues until the end of the attack, at first harsh and croupy, later looser with expectoration. The voice improves in character but is liable to become hoarse, especially toward evening. A fatal result, despite some authorities to the contrary, must be considered a rare event. In cases in which we have a fatal result it probably arises from an extension of the inflammatory process to the lungs, causing a capillary bronchitis or bronchopneumonia.

Treatment.

Treatment must be directed toward three conditions: (1) The acute inflammation of the subglottic mucous membrane; (2) the mild chronic inflammation of the same membrane; and (3), the nocturnal exacerbations.

Treatment of the Acute Process.—The child should be kept in a room at an even temperature of about 70° to 72° F. The atmosphere of the room should be moist. The bowels should be acted upon by small doses of calomel or hydrargyrum cum creta. The larynx, if the child is old enough and tractable, should be treated with a cleansing solution such as Dobell's, followed by the application of an astringent. These are to be applied by the ordinary nasal atomizer. A weak solution (one-half to one per cent.) of cocaine in an oily menstruum will accomplish much. Ipecac or tartar emetic in any form should not be used. The object to be kept in view is to help to clear the laryngeal mucous membrane and to aid its freer secretion. Muriate of ammonia or the carbonate is as efficient as any drug to accomplish this purpose. Due regard must be paid to the condition of the stomach in administering cough mixtures or the preparations of ammonia. If the stomach is in any way disturbed they should be abandoned. The use of opiates should be avoided, yet when, by loss of sleep or for other reason, a sedative becomes necessary, codeine or sufficient doses of Dover's powder should be administered. When, as is often the case, a diseased condition of the nasal passages is present, local treatment to these parts is of special importance. They should first be cleansed by the use of the nasal atomizer with one of the alkaline solutions already recommended, then sprayed

with a small amount of solution of cocaine, afterwards an astringent solution or a solution of nitrate of silver, five to ten grains to the ounce, should be applied to the nasal or naso-pharyngeal mucous membrane. If the confidence of the child can be obtained, this procedure may be carried out without insurmountable difficulties.

Treatment of the Chronic Condition.—We have oftenest, underlying the chronic condition, a cause such as enlargement of the faucial tonsils, the presence of the pharyngeal tonsil, or diseased conditions in the nasal passages. The treatment indicated is the removal of such causes. The general condition of the child, particularly the enlargement of the lymphatic glands of the throat and cervical regions, should be watched. Some form of iodine, probably best the iodide of iron, either alone or with cod-liver oil, is useful and should be continued over a long period of time. Hygienic details in regard to bathing, dress, and exercise are of the highest importance.

Treatment of the Nocturnal Exacerbations.—The older method, production of emesis, relieves the larynx, but its accomplishment by emetic drugs is liable to do more harm than good. The finger introduced into the fauces accomplishes the same purpose, if it seems necessary for immediate relief, with less disturbing effects. The first thing to do with a child suffering from the dyspnœa of a subglottic laryngitis is to put it into a bath at a temperature of about 100° F. If this measure fails to relieve, steam inhalations should be used. Hot applications over the larynx by means of a sponge or towel wrung out of hot water to which mustard has been added, have a derivative action. Blistering or leeching over the larynx probably accomplishes no purpose. Inhalations of a small amount of ether may relieve the dyspnœa when other remedies fail. The insertion of an intubation tube or the performance of tracheotomy is the resort when relief cannot be afforded by other means. A simple measure when proper instruments are not at hand is the passage of a flexible catheter through the larynx into the trachea. Such radical measures, however, are seldom called for in true subglottic laryngitis, but as the case may not at once be differentiated the symptoms must be treated as they arise. The treatment of the chronic condition which leads to the production of the disease is of the greatest importance.

Chronic Laryngitis.

Under this general term we describe three affections of the laryngeal mucous membrane, which, while they constitute distinct diseases from a clinical point of view, are grouped together on the ground that they are all essentially catarrhal processes. They are: First,

chronic catarrhal laryngitis; second, chronic subglottic laryngitis; and third, trachoma of the larynx, or chorditis tuberosa.

Chronic Catarrhal Laryngitis.

By this term we mean a chronic inflammation of the mucous membrane lining the laryngeal cavity, which is purely catarrhal in nature. This condition involves no special impairment of the ordinary functions of the larynx in the ordinary use of the voice. Many people undoubtedly suffer from this form of disease without experiencing any noticeable discomfort or inconvenience therefrom. With public speakers and singers the case is different, and for them it is essential that the organ should be in a state of absolute health.

Etiology.

It is doubtful if a simple uncomplicated chronic laryngitis is ever a primary disease. Certainly no case has come under my own observation in which development of the morbid process could not be traced to some diseased condition of the air passages above. This method of development is produced by the interference with the normal respiratory process which a diseased condition of the nasal passages entails. Where the respiratory function of the turbinated bodies is interfered with, the inspired air may reach the larynx through the mouth; it is in such case not warmed, moistened, or filtered, as it would be in its passage through the nose, and so leads to the development of an inflammatory process in the laryngeal mucous membrane. The presence of the pharyngeal tonsil, as may be seen, leads in a similar way to disturbances in the larynx. In an atrophic rhinitis the turbinated bodies are more or less destroyed and the current of air is neither moistened nor cleansed in its passage through the nose. Between the ages of five and fifteen a chronic laryngitis is generally the result of the presence of the pharyngeal tonsil. Between the ages of fifteen and forty we find catarrhal diseases oftener dependent upon some obstructive lesion in the nasal passages. A chronic laryngitis at this age depends, in the large majority of cases, upon an irregularity of the septum or a hypertrophic rhinitis. After the age of forty we less often recognize an hypertrophy of the turbinated bodies as the result of a deflected septum, but seek the source in a chronic naso-pharyngitis.

The above classification, while perhaps not absolutely correct, certainly applies to a large majority of cases. Habits of life, occupation, and the excessive use of the voice or its improper use, etc., are commonly ascribed causes of chronic laryngeal catarrh. While this may possibly exert some influence, I think some morbid con-

dition such as described will be found the active predisposing cause. Those occupations which involve the inhalation of vitiated or dust-laden atmosphere may have a marked influence in producing laryngeal inflammation, yet even in these cases the influence of such conditions may be overestimated. The mucous membrane in the upper air tract is very tolerant, and Nature has provided an admirable method of cleansing the inspired air in such a way that but little of its impurities reaches the larynx. Moreover, I doubt if the mere lodgment of dust or other particles on the mucous surface is sufficient as a rule to excite a true inflammatory process. The use of alcohol in moderation has but little influence upon the larynx. Its excessive use is attended by a diffuse general inflammation through both the naso-pharynx and the oro-pharynx, this condition setting up eventually a chronic laryngeal catarrh. The use of tobacco is generally unattended by disturbances of the laryngeal mucous membrane; in certain individuals, however, any excess is invariably followed by a laryngeal hyperæmia. An elongated uvula frequently gives rise to faucial irritation, but rarely to well-developed laryngitis. All catarrhal diseases of the upper air tract are more common in males than in females, and the same statement applies to the larynx. It occurs at all ages but most frequently in adult life and middle age. The disease is not of a serious character, and but a small proportion of the cases present themselves for relief at the hands of the surgeon.

Pathology.

The changes consist mainly in dilatation of the blood-vessels, together with a certain degree of hyperplasia, resulting in an organic thickening of the mucous membrane. These are seen in the whole membrane lining the larynx, particularly in the ventricular bands and over the arytenoid cartilages. There is an increased vascularity in the vocal cords, but hyperplasia is rare. Different authors describe a form of disease as "chronic glandular laryngitis," in which the inflammatory process is regarded as an extension of follicular pharyngitis or "clergyman's sore throat." Cases in which the morbid process is confined to the muciparous glands must be rare. Where the involvement of the lymphatics is the chief feature I should regard it as an evidence of a systemic condition. The changes chiefly consist in an increase of the epithelial and connective-tissue elements of the mucous membrane.

Symptomatology.

The most striking symptom in this disease is a change in the voice, this becoming husky and hoarse. It may not be especially

noticeable in the ordinary speaking voice, but it is remarked upon singing or upon slight exercise or prolonged use. Cough may be present. There is a disposition to clear the throat of mucus. The disease is usually dependent upon some affection of the nose or nasopharynx, and in consequence of this, laryngitis or tracheitis, and in some instances bronchitis, may be set up. If cough with expectoration be present the disease has probably extended beyond the larynx. The condition of the voice is worse in the morning, but after clearing away the accumulation of mucus it may be normally clear. As the day progresses it is apt to grow weaker and husky. Its use in singing or continued speaking or reading brings on an impairment both in strength and volume. It is liable to become completely lost after any excessive effort and a sense of irritation or pain may be produced. In singers and in public speakers the impairment is not only of more serious importance but it is also more frequently and markedly seen in them. The whole register of the voice is involved, but particularly the higher tones. The feeling of fatigue and pain may be accounted for by the increased effort of the laryngeal muscles required to effect a proper tension of the cords under these unfavorable circumstances. We have, besides, from the increased effort more frequent and shorter breathing, often through the mouth, with the result of an abnormal drying of the mucous membrane.

Diagnosis.

The disease should be easily recognized by the subjective symptoms. Impairment of the voice is an indication of a diseased condition of the laryngeal membrane; a definite diagnosis must, however, depend upon a laryngoscopic examination to eliminate paralysis, the presence of tumors, an ulcerative or exudative affection. The absence of cough is an indication that the parts below are not involved. The laryngoscopic examination shows the ordinary appearances of a chronic inflammatory process. The mucous membrane presents a reddened, injected, and a more or less swollen appearance. This is seen best over the ventricular bands and the arytenoid commissure. Some grayish mucus may be seen covering the mucous membrane, but as a rule an excessive secretion is not apparent. These appearances are usually evenly diffused and symmetrical. Brown and Mackenzie speak of the signs of hyperæmia and thickening in some instances as being unilateral. I should regard this as a suspicious circumstance and one to suggest the existence of such an affection as an ulcerative or malignant disease, unless a foreign body were present or there were some other obvious cause. The mucous membrane covering the cords is slightly swollen, the cords

presenting in place of their usual whiteness a somewhat grayish discoloration. The cords may show certain irregularities on the surface or on the edges, the result of the epithelial proliferation. This is most marked in the neighborhood of the vocal processes of the arytenoid cartilages. Minute blood-vessels are occasionally seen standing out in relief upon the white fibrous tissue of the cord itself; more commonly we have a grayish or slightly reddened, uniform discoloration. Adduction of the cords is less perfectly performed than in health; this is especially noticeable when the arytenoid commissure is swollen and when there is marked epithelial proliferation at its anterior face, constituting an almost wart-like appearance in this neighborhood. In these cases there is a mechanical interference with the approximation of the arytenoids as well as with the inward rotation of the vocal processes.

Prognosis.

A spontaneous cure of a chronic laryngitis, or any effort on the part of Nature to accomplish a restoration of the parts to a state of health, is not evident. The dependence of the condition upon a previously existing disease of the parts above perhaps precludes the possibility of this occurrence. The disease runs an indefinite course, and its symptoms persist so long as the morbid affection of the passages above, which is responsible for it, endures. The symptoms may be aggravated by such special circumstances as have been described. They usually persist in much the same degree except when thus aggravated. Repeated acute exacerbations due to an exposure or other cause take place. A point of interest in the prognosis is the possibility of the development of more serious trouble, such as phthisis of the lungs, or of a benign or malignant tumor. A chronic laryngitis is one of the most frequent of affections, whereas tumors of the larynx are undoubtedly rare. It is, however, somewhat unusual to meet with a laryngeal growth in a patient who does not at the same time show evidences of chronic catarrhal disease of the passages above. A catarrhal inflammation must be regarded as an active factor in the causation of laryngeal neoplasms. While the condition of laryngitis may be the result of a growth, the clinical history usually shows that a diseased condition of the air passages existed before the development of the tumor. I do not think that the teachings of clinical experience, or our knowledge of pathological processes, give any support to the view that a simple catarrhal process in the larynx is either an active or a predisposing cause of a cancerous growth. I know of no clinical observation which justifies us in regarding tubercular disease as even a remote danger in a sim-

ple catarrhal process. When we find a tubercular affection following a chronic laryngitis it is exceedingly rare that we cannot trace it to a more powerful and direct predisposing cause than a simple catarrhal inflammation of the laryngeal mucous membrane. Indeed, when we consider how totally different the two processes are, I think we are justified in the conclusion that the milder process is not even a predisposing cause of the graver disease. An element of prognosis is the curability of the catarrhal disease. The general opinion is that the voice is liable to remain weak and hoarse even after proper treatment of the morbid conditions. This I regard as sufficient evidence than the disease had not been cured. By a cure I mean not only that of the local morbid process, but of the more important conditions in the air passages above which have been active in its causation. With the removal of all diseased conditions the voice should be restored to its fullest functions.

Treatment.

The first and most common indication in the treatment consists in an endeavor to restore the respiratory passages above to a condition of health. If there exists a deflected septum, hypertrophy of a turbinated body, a chronic naso-pharyngeal catarrh, or enlargement of the pharyngeal, faucial, or lingual tonsils, the appropriate treatment for one or several of these conditions must be instituted. The laryngeal disorder often subsides spontaneously with the removal of the active cause of the disease. Its progress toward cure may be facilitated and hastened by remedies applied directly to the diseased membrane in the larynx. An atomized fluid is generally to be preferred to the use of the brush or sponge, because of the liability of the last to do harm even in the hands of the well-trained manipulator. In the use of the atomized fluid either from some simple hand-bulb instrument or from one used with compressed air, the tongue should be well protruded, the point of the instrument being passed well backwards over the crest of the epiglottis. The use of the mild astringents I believe is more efficient than that of the stronger ones. The indications for caustics or agents to destroy tissue do not exist in this disease. I regard nitrate of silver as probably the best of all local astringents. As a rule a ten-grain solution is of sufficient strength. Other drugs (and a change from one to another is sometimes desirable) are, as given in the order of preference: Argenti nitras, gr. v. to xx. to the ounce; zinci sulphas, gr. v. to xx. to the ounce; zinci chloridum, gr. ij. to vi. to the ounce; liquor ferri persulphatis, ♏ x. to xxx. to the ounce; cupri sulphas, gr. iij. to x. to the ounce. We thus give decided preference to the use of the mineral astringents. I have never seen any special good accomplished

in this disease by the vegetable astringents such as tannic and gallic acids or drugs of this order. The same, I think, can be said of iodine and its preparations, carbolic acid, creosote, etc.

Cold inhalations of fluids atomized by compressed air form a rather attractive method of treating this affection, and their use is attended with a certain amount of comfort, and yet as a rule, I think, their efficacy is rather over-estimated, unless the disease extends somewhat into the tracheal and bronchial tubes. The old apparatus of Lewin for the inhalation of nascent muriate of ammonia is an ingenious affair, and its use gives a certain amount of relief, but the direct action of ammonia on the mucous membrane is probaby somewhat feeble. Its efficacy is increased undoubtedly by adding to the ammonia solution a small amount of creosote, or carbolic acid, oil of turpentine, oil of peppermint, or oil of cloves, or other similar remedy. Inhalations by means of a steam atomizer, as already mentioned, possess the objectionable feature of causing a certain amount of relaxation of the membrane. We have already spoken of the use of throat lozenges. Their action in these affections is necessarily very limited except as affording a certain amount of relief to the congested membrane by the use of those containing ammonia, cubebs, and other stimulating drugs. But, of course, it is idle to suppose that any portion of the remedial agent which they contain comes in contact with the mucous membrane of the larynx. We thus find that our topical measures in the treatment of laryngeal catarrh consist mainly in the direct application to the diseased mucous membrane of mineral astringents in the form of spray, and the question of local therapeutics becomes therefore an exceedingly simple matter. This I believe is as it should be, for undoubtedly the local treatment of the laryngeal membrane is a far simpler matter than we are led to suppose in consulting the literature on the subject, which we often find somewhat prolix in the discussion of minute and unimportant points, while the important feature of the whole matter is overlooked—viz., that a simple catarrhal laryngitis is always, probably, secondary to a diseased condition of the air passages above, and that our success in the treatment of any given case is based upon the recognition of this fact and the adoption of measures for the correction, not only of the diseased condition of the larynx itself, but of the whole upper air tract. These topical applications to the larynx should be regulated as to their frequency somewhat according to the severity of the attack and the immediate circumstances of the case. Occasionally we meet with instances in which the importance of voice restoration demands that the treatment be repeated daily or possibly oftener; as a rule, however, two or three treatments a week will suffice as long as careful attention

is directed to controlling the disease in the passages above. The strength of the solution used should also be regulated according to the severity of the attack and the results obtained. As the morbid process diminishes in activity the larynx, of course, becomes less tolerant of strong solutions. The use of caustics in the larynx very early in a simple catarrhal process is never indicated, but not rarely we meet with cases of long-standing chronic laryngitis which have taken on a form of pachydermia in which there is notable hypertrophy of tissue at the posterior insertion of the cords and on the anterior surface of the arytenoid commissure, which gives the appearance of warty excrescences. In cases of this kind simple astringents are not sufficient to produce resolution, the indications then point toward the use of some destructive agent. It is not a matter of so much importance what agent we use as that the application shall be made with sufficient dexterity that the sound tissues are not injured. My own preferences are for the use of chromic acid fused on the end of a slender hooded applicator; although nitrate of silver in solid stick or the galvano-cautery may be used. If the case be that of a public speaker or singer too much importance cannot be attached to the absolute necessity of rest to the organ, referring, of course, to vocal rest, for the respiratory movement of the larynx, it is unnecessary to state, cannot be controlled. Furthermore, these movements probably in no way interfere with the result of an inflammatory process, while we can easily understand that the use of the voice in phonation will not only seriously retard the cure, but markedly interfere with the efficacy of the topical agents which are applied to the diseased parts. I scarcely need repeat here what I have frequently urged in former writings, viz., that wherever a catarrhal process exists certain hygienic measures not only greatly facilitate the prompt resolution of the disease, but also materially aid the local measures adopted. I refer to the proper regulation of the dress and the promotion of healthy functional activity of the circulation by the daily use of cold sponging, not only about the head and neck, but over the whole body. There is no single measure which I regard as of more importance in the treatment of catarrhal disease, whether of the larynx or other portions of the air tract, than the application of cold water to the skin, followed by a vigorous rubbing with a coarse dry towel. We thus not only promote and stimulate a healthy circulation (and when we refer to the circulation we mean the capillary circulation of the skin), but we also promote a healthy activity of the other important function of the skin, viz., that of perspiration by which bodily heat is regulated.

There are two indulgences which in the minds of the laity are

regarded as exercising an especially pernicious action upon the mucous membrane of the upper air tract. These are the use of tobacco and the use of alcohol. The pernicious action of tobacco I regard as one which is greatly over-estimated. The injurious effect of tobacco lies in the absorption of nicotine—a drug which possesses a peculiarly toxic action on the nervous system and especially on the pneumogastric nerve. This is accomplished by its absorption and not by the direct action of the smoke upon the mucous membrane; hence while, of course, the excessive use of tobacco is in every way to be discouraged, or even interdicted, its moderate use is probably not especially injurious to the upper air tract. I am confident from a somewhat extensive experience that the direct action of tobacco smoke upon the mucous membrane of the upper air tract is not especially injurious. It may for the time being irritate an existing catarrhal process, but that it produces any permanent effects I do not believe. The use of alcohol, on the other hand, I consider by far the most pernicious, as regards the upper air tract, of all the habits which lead to over-indulgence. Its action is somewhat indirect as regards the larynx, for, according to my observations, its first effect is to produce a chronic gastric catarrh; secondly, a chronic naso-pharyngitis; and, finally, if this persists for any length of time, the whole of the upper air tract including the larynx becomes deeply congested and relaxed, and a condition of chronic catarrhal inflammation results which is absolutely not amenable to any treatment, so far as my experience teaches me, until the indulgence is altogether interdicted. There are a number of nerve tonics such as coca wine and hypophosphites and other drugs of similar character which have obtained a certain vogue as voice tonics. That these possess any direct action upon the vocal organs, I do not believe. Their efficacy is merely a recognition of the fact that any general depression of the nervous system reacts directly upon the larynx, and that a correction or a toning up of the general symptom thus has an effect, although not direct, upon the vocal apparatus. In much the same way there are certain mineral waters which are supposed to be of value in the treatment of laryngeal catarrh, whose action is not directly upon the larynx itself but rather upon the naso-pharynx, which, as we know, is in very close sympathy with the intestinal tract, and any improvement in the laryngeal affection which is the result of their use must be attributed to their favorable action upon the digestive apparatus. Of course, we must not overlook the fact that the rest and regular hours which result from a few weeks spent at a mineral spa are of special service in giving tone to the system and thus indirectly have a favorable influence upon catarrhal disorders of the upper air tract.

Chronic Subglottic Laryngitis.

This form of laryngitis, while fortunately somewhat rare, is one of far greater importance than that which we have just discussed, in that the tissues below the vocal cords become the seat of hyperplastic changes whereby the lumen of the larynx is seriously encroached upon, and hence dyspnœic symptoms of more or less well-marked character may ensue. It was first somewhat generally alluded to by Rokitansky in 1852, while the first detailed report of a case was made by Czermak in 1863. Still later Türck quotes two cases under the name of chronic œdema; Scheff wrote on the subject as hypertrophy of the cords, while Gerhardt gives it the name of chorditis vocalis inferior hypertrophica. That it was undoubtedly overlooked in former years in many instances is shown by the fact that Mackenzie reported twenty-three cases which came under his own observation.

Etiology.

It belongs essentially to the early period of life and usually occurs between the ages of fifteen and twenty-five, although Catti has observed a case at forty-nine. It is more commonly met with among females than males, thus departing from the ordinary rule as regards simple catarrhal cases. This would seem to indicate that the affection is dependent upon a systemic diathesis. Moreover, in a majority of the cases reported the individuals are said to have shown notable evidences of the scrofulous habit. We have already stated that we regard this to be due to morbid changes in the lymphatic tissues which are found in the mucous membrane of the larynx immediately below the vocal cords; and furthermore, we took the ground that this was to be regarded as evidence of lymphatism—a systemic condition which, while not constituting scrofula, is very closely akin to it, and a morbid condition which, if carried one step farther, would result in true scrofula. The inference of course is that the lymphatic habit of childhood may be an actively predisposing cause, if not the direct cause of this form of laryngitis as met with in later life. In several of the cases which have come under my own observation this lymphatic habit was notable and was shown by the existence of large lymphatic masses in the parts above in the form of faucial and lingual tonsils. Furthermore, the subglottic hyperplasia in each of these cases was notably ameliorated either by the removal of the lymphatic masses above, or by the administration of large doses of the iodide of iron to correct the systemic condition. Schroetter enumerates somewhat vaguely among the causes of the disease, tuberculosis,

typhoid fever, rhinoscleroma, and perichondritis. That typhus and typhoid fevers may undoubtedly give rise to the disease clinical experience shows; but that form of laryngeal disorder which is most liable to ensue from these exanthemata is perichondritis. Tuberculosis and perichondritis constitute morbid processes which bear no relation either direct or indirect to lymphatic disease. The same can also be said of syphilis. There may be also a certain relation between rhinoscleroma and this form of laryngitis, but it is by no means notable. It is possible that a simple idiopathic inflammatory process in this region which is not attendant upon or connected with any pronounced systemic condition may give rise to a chronic subglottic laryngitis. In the large majority of instances, however, I think we must look for some diathetic state to explain the fact of a simple catarrhal process resulting in such marked hyperplastic changes as to notably encroach upon the breathing space.

Pathology.

We have absolutely no data to go upon in determining what pathological changes constitute this form of laryngitis, hence any suggestions we may make will be purely speculative. We content ourselves, therefore, with the suggestions already made in discussing the etiology of the disease—viz., that it is probably in most instances a form of lymphatic hypertrophy. The only post-mortem examination which has been made, as far as I know, in a case which was supposed to be subglottic laryngitis is that of Bandler. In this case the disease began in the nose and successively invaded the air passages below as far as the lower end of the trachea. The patient succumbed to an attack of pneumonia, and the post-mortem examination revealed the morbid condition to be that of rhinoscleroma. This would lend weight to the suggestion that many of the cases reported as instances of subglottic laryngitis are probably true rhinoscleroma.

Symptomatology.

The onset of the disease is liable to be somewhat insidious and is characterized by a slowly developing but progressive impairment of the voice, which finally results in a complete aphonia, this aphonia being due not necessarily to an involvement of the vocal cords, but rather to an infiltration of tissues which prevents their approximation. Cough is not present in the early stage of the affection, although it may become a very prominent and an exceedingly distressing symptom later on, especially on the occurrence of exacerbations or acute attacks which result from an ordinary exposure to cold. During these attacks dyspnœa is liable to set in, and a more

or less free secretion is poured out into the parts; this is apt to be thick, viscid, and tenacious, hence in the effort to dislodge and expel it the cough becomes an exceedingly persistent and distressing symptom, and takes on a harsh, metallic, croupy sound. The dyspnœa is more or less well marked according to the extent of the swelling, and is both expiratory and inspiratory. As the lumen of the larynx becomes more encroached upon, the breathing becomes somewhat labored and is marked by an inspiratory and expiratory stridor. As the disease progresses the two symptoms which become most prominent after the aphonia are the cough and dyspnœa, both of which tend to increase. In two of the cases which came under my observation the cough was not only persistent, but occurred in paroxysms and assumed a harsh clangor which we recognize as characteristic of laryngeal chorea. It becomes almost a bark or a succession of barks, suggesting whooping-cough. As the symptoms develop the dyspnœa becomes still more marked, and finally tracheotomy is liable to become necessary unless other simple measures have availed to give relief.

Diagnosis.

The harsh metallic character of the cough in connection with the dyspnœic symptoms and the duration of the disease would serve to suggest the location and form of the morbid process, although a direct laryngoscopic inspection alone of the part would serve to make the diagnosis certain. This will show the mucous membrane of the upper portion of the larynx presenting the ordinary appearances of a mild, chronic laryngeal catarrh. The vocal cords themselves are liable to be somewhat thickened, and their mucous membrane injected. The characteristic conditions which constitute the disease will be seen particularly below the vocal cords in the two rounded or oval-shaped masses which project from the sides of the subglottic portion of the larynx beyond the inner margin of the vocal cords. These masses are either grayish, opaque, or of slightly reddish tinge, or they may be deeply injected, presenting the appearances of an ordinary mucous membrane in a state of chronic inflammation. They have a solid, unresisting aspect as of a true hyperplastic or hypertrophic process. They are covered with a thin, scanty secretion, the normal secreting apparatus having undoubtedly become to a certain extent atrophied by the encroachment of the hyperplasia. If an acute exacerbation is present at the time of examination the membrane will have the appearance of an ordinary acute inflammation extending to the subglottic larynx. The movements of the cords in abduction are not noticeably impaired, while adduction may be seriously interfered with by the thickened tissue. The appearances are illustrated in

Fig. 51. The only affection with which this need be confounded is perichondritis; this latter disease, however, instead of presenting a bilateral and symmetrical tumefaction presents one which is irregular in outline, asymmetrical, and almost invariably unilateral.

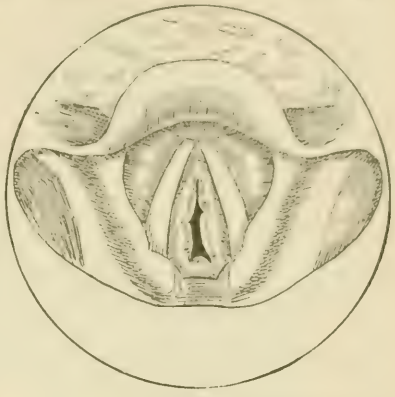


Fig. 51.—Chronic Subglottic Laryngitis.

Prognosis.

In a few of the reported cases where the disease was due to an attack of typhus fever, the symptoms developed very rapidly and tracheotomy became necessary at the end of one or two months. In most of the reported cases the cause of the disease has been obscure. These cases were marked by a much slower and more insidious development, and the dyspnœa did not appear until from nine to eighteen months after the first manifestations of the disease. I know of no case, either in my own experience or in literature, in which spontaneous resolution has occurred. In the large majority of reported cases tracheotomy has been necessary, and when this was done the wearing of a tube became permanent. In one or two cases the insertion of the tube seemed to be followed by a certain amount of reduction of the tumefaction, but in no instance was this sufficient to admit of the permanent removal of the tube. While, therefore, in a certain number of cases remedial measures have served to eradicate the disease permanently, in the majority of reported instances the disease has proven incurable. What our prognosis should be, therefore, in any given case becomes somewhat of a problem. If the disease occurs in connection with rhinoscleroma of the nasal passages the prognosis is of course very unfavorable. If, on the other hand, it seems clearly a case of lymphatism and occurs in connection with faucial or pharyngeal tonsils, we are justified in the view that the graver symptoms will not ensue, and probably our therapeutic measures will serve to eradicate the disease.

Treatment.

From what we have already said it will be understood that we have practically to do with subglottic organic stricture of the larynx. In the majority of the cases, either as the result of the failure of local measures to arrest the disease or in consequence of unimpeded development, tracheotomy becomes imperative. This was done in nine

of Mackenzie's twenty-three cases. No simple topical applications seem to have been of any avail. Bergmann performed thyrotomy successfully and destroyed the thickened tissue with the Paquelin cautery, whilst Sokolowski attained similar success by excising the thickened tissue after opening the larynx. Voltolini advises the use of the galvano-cautery applied through the natural passages, while Mackenzie advises scarification. Czermak's case (the first reported) was treated with dilating by bougies passed directly through the point of stenosis. Although this method of treatment seems to have been a failure in Czermak's case, it has proven, perhaps, more successful than any other measure in the majority of cases reported subsequently, although instead of using the solid bougie, hollow bougies or O'Dwyer's tubes were used. In this way dilatation may be maintained for a considerable period of time. In most of the successful instances reported the result was absorption of tissue, although in one of Catti's cases the diseased tissue sloughed away leaving a permanent opening sufficient for breathing. A successful result was obtained in most cases in from eight to fifteen months of treatment. No case of this disease has come under my own observation in which the morbid process had resulted in sufficient stenosis to cause grave dyspnœa. Moreover, in all the cases evidences of the lymphatic habit were sufficiently notable to warrant the conclusion that the stricture was due to lymphatic hyperplasia. In these cases, as already intimated, the faucial and pharyngeal tonsils and the lymphatic tissue at the base of the tongue, where present, were freely excised and the patients put immediately upon large doses of iodide of iron; the result in all cases but one was eminently satisfactory; in the one case which failed of complete relief notable improvement was secured. My own clinical experience has taught me that the systemic condition of lymphatism yields more readily under the administration of iodide of iron than of any of the other preparations of iodine. I am sure that in many instances failure of this drug has been due to the fact that it has been administered in far too small doses. To get its best effect it should be given in at least from ten to twenty grain doses in adult life and proportionately for children.

Trachoma of the Larynx or Chorditis Tuberosa.

This disease was first described by Türck under the name of chorditis tuberosa, and later has been frequently discussed under the name of singers' nodes; it consists in the development on one or both of the vocal cords of small nodules or tuberosities midway between the vocal processes and the anterior insertion of the cords. It is

occasionally classified under the heading of one of the laryngeal neoplasms. From a clinical point of view it seems more appropriate to regard it as one of the forms of laryngitis, in that it ordinarily accompanies a chronic laryngeal catarrh and is one of its late developments. It makes its appearances as a small, rounded projection which is sessile in character, standing out from the free border of the vocal cord and showing itself distinctly in profile under a laryngoscopic examination (see Fig. 52). It appears to be practically a tumefaction of the fibrous tissue of the cord protruding from it, the same uniform color showing through the very thin mucous membrane. After it has developed it seems to remain stationary and shows no tendency to increase in size. It may develop on a single cord to be subsequently followed by a similar process on the opposite cord in exactly the same situation, or it may develop on both cords simultaneously. In my experience it only occurs as the result of an attempt to use the highest powers of the larynx when it is in a condition of chronic catarrhal inflammation, to exert an effort which can be accomplished with impunity only when the larynx is in a state of absolute health. In the production of the singing voice in the lower and middle register, as we know, the notes are produced by vibrations of the vocal cords in their full length, the higher pitch being secured with each additional note by greater tension of the vocal cords. When we have reached the top or middle register the vocal cords seem to have reached their limit of tension, hence still higher notes must be produced by shortening the vibrating cords. This is accomplished by gradually shortening the chink of the glottis by bringing into apposition the edges of the posterior portion of the cord, until, finally, in the production of the upper notes of the head register the vibrations are confined only to the anterior third of the vocal cords. How this was accomplished was always somewhat of a mystery until Madame Seiler showed the existence of a small, slender, rod-like fibroid cartilage embedded in the vocal cord and extending from the vocal process to the junction of the middle and anterior third of the cord. It is by means of this fibro-cartilage that the posterior portion of the cords are held in apposition, and the chink of the glottis in the high notes of the register is thus composed only of the anterior third of the two

Fig. 52.—Trachoma of Right Vocal Cord.

cords. It is at the posterior termination of this chink that these nodes or singers' nodes appear; hence, since we know that the very highest muscular powers of the larynx are required for the production of these high notes, I am disposed to think that these nodes are the result of this excessive effort in holding the posterior portions of the cords in apposition during the production of these high notes in a larynx which is the seat of an inflammatory process. If this theory is not correct, it is certainly a curious fact that these singers' nodes always develop at exactly the same point on each cord, and that point is the posterior termination of the glottis in the upper register. Türck made the statement in first describing the disease "that these nodes were due to hypertrophy of the gland structures," but, as we know, the mucous membrane covering the vocal cords has no muciparous glands. The pathological lesion is probably somewhat analogous to pachydermia with the addition of a certain amount of connective-tissue hypertrophy. Wedel has also found a number of nuclei embedded in the tissues, which seems to indicate the existence of an inflammatory process.

Symptoms.

The symptoms to which the condition gives rise are chiefly functional. There is no pain, no cough, and no reflex symptoms are present. There is simply hoarseness resulting from a mechanical interference with a perfect approximation and free vibration of the vocal cords. This hoarseness is noticeable in the conversational voice as well as in the singing voice, but of course it is especially noticeable in the singing voice, and the taking of high notes is rendered almost impossible, while all the notes of the singing register are markedly impaired.

Diagnosis.

The diagnosis is easily made by a laryngoscopic examination, the mirror revealing, on one or both cords, a small, whitish-gray nodule, always in the same location, and especially prominent on attempted phonation.

Treatment.

The treatment consists mainly in topical applications to correct the chronic laryngitis which accompanies the affection. If the affection has lasted but a short time, or, in other words, the hyperplastic tissue is not organized, I think it is not infrequently possible to secure complete resolution by the application of a strong solution of nitrate of silver; this should be applied directly to the larynx by

means of cotton wrapped on a bent applicator. These applications should be made at least three times a week, or, better still, every day, while at the same time the use of the voice should be absolutely interdicted in singing, and as far as possible in ordinary conversation. Of course, all thought of continued singing practice must be abandoned until the disease is completely arrested. If this measure fails to give entire relief, I think the next should consist of the galvano-cautery, the larynx being thoroughly anæsthetized by the application of a ten or twenty per cent. solution of cocaine. A fine-pointed electrode should be inserted, just touching the point of the nodule while the cords are in a state of approximation, and the current be turned on and almost immediately shut off again, thus avoiding any possible risk of too much destruction of tissue. Too much importance in these cases cannot be attached to thoroughly eradicating the cause of the chronic catarrhal process. I do not think singers' nodes develop in a healthy larynx, and furthermore, I do not think a catarrhal laryngitis exists except as the result of disease above; hence if the nodes are destroyed they are liable to return again unless the exciting cause has been thoroughly abolished.

Laryngitis Sicca.

The term laryngitis sicca is applied to a catarrhal inflammation of the larynx in which there is a deficiency of secretion, and in which crusts are formed, lodge upon, and become adherent to the mucous membrane. Rühle was the first to refer to this condition in 1861.

Etiology.

I do not know of any reported case unconnected with a diseased condition of the parts above. In most cases it has been found to coexist with an atrophic rhinitis; or there might be an atrophic condition of the naso-pharynx or the oro-pharynx. In spite of this apparent indication that the laryngeal affection is symptomatic, we believe that some diseased condition must exist in the mucous membrane of the larynx leading to a diminution of or interference with its normal secretion, because atrophic processes of the nasal passages are of frequent occurrence, while laryngitis sicca is rare.

It has been suggested that the atrophic process is an extension from the parts above. I regard it as an almost invariable rule that catarrhal affections do not extend by continuity of tissue from the naso-pharynx into the larynx.

It is probable that the direct cause of the disease is an abnormal condition of the laryngeal mucous membrane causing functional dis-

turbance or destruction of the muciparous glands, with the secretion of a thick, inspissated mucus which forms crusts upon the membrane.

The inhalation of an abnormally dry atmosphere notably aggravates the symptoms of this disease.

Pathology.

Loewenberg's ozæna diplococcus has been found in the crusts, but we regard this as a concomitant circumstance, not a cause.

As a rule, the crusts form in the subglottic portion of the larynx only, which shows that the condition is not entirely symptomatic of a disease process above.

The name "laryngo-tracheal ozæna" apparently indicates involvement of the trachea. The crusts are found below the cricoid ring, but are probably masses of inspissated mucus, whose origin is the subglottic larynx.

Symptomatology.

The comparative quietude of the glottis during the night allows of a more rapid crust formation than during the day, causing distressing symptoms upon awakening such as total or partial aphonia and great irritation. Marked dyspnœa may occur from encroachment on the breathing space. Voluntary efforts to clear the throat at this time may bring relief, but the cough, discomfort, and injury to the voice usually persist during the day. The expelled masses are greenish-yellow, but are smaller than those discharged in atrophic rhinitis. The fetid breath, which is a constant symptom, is detected in the oral expiration, while that of atrophic rhinitis is more evident in nasal expiration. The expelled masses are often discolored with blood showing that the mucous membrane is eroded.

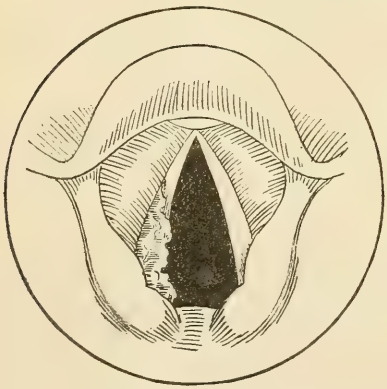


Fig. 53.—Crust Lying in the Ventricle in Laryngitis Sicca.

Diagnosis.

While the subjective symptoms are sufficiently marked to allow of a fairly correct diagnosis, a certain conclusion can be reached only by the use of the laryngoscope, by which means we can see the greenish-gray crusts adhering to the subglottic mucous membrane and projecting into the lumen. Rarely, thin flocculi of inspissated muco-pus, or a dry crust may be found above the vocal cords (Fig. 53), adherent to the ventri-

cular bands or to the arytenoid commissure; in these cases the activity of the morbid process is less. As a rule, however, the disease is confined mainly to the subglottic portion.

Course and Prognosis.

The disease is a chronic one, and it is only under treatment that it shows signs of improvement.

Should our view be correct that an atrophy of the glandular or secreting structures constitutes the essential lesion, the disease, unless treated early, would appear to be somewhat intractable. The presence of other morbid conditions modifies the prognosis; a coexisting incurable atrophic rhinitis would permit only of an amelioration of the symptoms, while the presence of curable affections of the nasal passages would give a favorable outlook.

Treatment.

Secretions appearing on the surface of the mucous membrane are fluid, even at an advanced stage of atrophy, and subsequent changes alone can convert them into a dry crust. There must be a morbid condition of the passages above, otherwise the inspired air would be sufficiently moist to prevent drying up of the laryngeal secretion. A healthy condition of the mucosa of the upper air tract is therefore the first desideratum. Any obstruction in the nasal passages should be removed, atrophic rhinitis appropriately treated, and any morbid condition of the naso-pharynx reduced.

Accumulated crusts and inspissated mucus should be removed as thoroughly as possible without too great irritation of the parts. The use of a brush or probang will usually be sufficient, while in mild cases the laryngeal atomizer will soften the crusts that they can be voluntarily expelled. Any of the usual alkaline and antiseptic solutions may be employed, such as Dobell's solution.

After cleansing of the parts, we may apply directly, by means of a sponge or a cotton pledget, one of the following substances: Argenti nitras, gr. x. to xx. to the ounce; zinci sulphocarbolas, gr. xv. to the ounce; zinci chloridum, gr. x. to xv. to the ounce; acidum tannicum, gr. xx. to the ounce; acidum lacticum, ℥ ss. to the ounce; tinctura iodi composita, ℥ i. to iij. to the ounce. These are given in the order of preference. As a rule, the part to be treated is the portion of the larynx below the glottis. A quick movement during inspiration will insure the easy passage of brush or probang. The laryngoscopic mirror must be used.

If the symptoms are distressing, the patient should between treatments use a cleansing solution, three or four times daily, by means of an atomizer. A solution of lactic acid, two or three drops to the ounce, in an inhalator has been known to give more permanent results than nitrate of silver locally applied. Carbolic acid, creosote, oil of tar, acetic acid, etc., may be used. Internal medication is not usually indicated in this purely local disease, but creosote internally has been said to give good results.

Acute Phlegmonous Laryngitis, or Œdematous Laryngitis.

That variety of inflammation of the larynx which is distinguished by great swelling of the parts from serous infiltration has been called œdematous laryngitis. Œdema of the glottis we consider an erroneous term, since the glottis proper is usually not affected, or much less so than the parts above.

The utmost confusion in classification appears to exist among writers; but it seems to me that a simple classification of such diseases of the larynx as are accompanied by œdema can be made by dividing them into "phlegmonous laryngitis" and "symptomatic œdema," or simply œdema of the larynx, the latter term being used only for passive, non-inflammatory œdema; where there is acute inflammation of the mucosa of the larynx occurring idiopathically or as a complicating lesion of an acute, unusually active, deeply seated inflammatory process of neighboring parts, which causes great tumefaction from serous infiltration, the term phlegmonous laryngitis or acute submucous laryngitis is applied.

Etiology.

An exposure to cold is practically the only cause known in this affection, the primary, essential lesion of which is of an inflammatory nature. The local condition is really an acute cellulitis.

Sometimes the attack appears to be the result of a mild acute laryngeal catarrh, but it may occur where there is absolutely no affection of the air passages. Excessive use or strain of the voice is believed by some to be a cause of the disease. Men are more subject to it than women, the majority of cases occurring in persons between twenty and thirty years of age, although some have been noted at nine weeks and some at eighty years.

Investigations by Hoffman and Lestier have shown that the affection is comparatively rare. Secondary phlegmonous or submucous laryngitis occurs more frequently, being caused by quinsy, abscess of

the neck, glossitis, acute, probably erysipelatous, pharyngitis, and iodism.

Fehleisen's coccus having been found in cases of phlegmonous laryngitis, the theory that this disease, which is so frequently associated with erysipelas, is in reality often true erysipelas of the larynx, is undoubtedly correct. Whatever its origin, the development, course, symptoms, and treatment of the disease remain the same. It may occur during the course of typhoid fever, variola, diphtheria, croup, typhus, and the exanthemata. The impaction of a foreign body in the larynx, the passage of one into the œsophagus, the swallowing of corrosive poisons, even the inhalation of acrid vapors and of hot steam, may by traumatism cause the disease.

Pathology.

As we have previously stated, the process and consequently the morbid changes are inflammatory, but there is a greater activity than is usual in inflammations. From turgescence of the blood-vessels with serous transudation, the membrane, especially wherever it is most loosely attached, becomes enormously swollen and tense. The vocal cords are not frequently involved, the subglottic portion of the larynx is rarely affected, but the aryepiglottic folds, the ventricular bands, and the posterior surface of the epiglottis are favorite seats of the disorder.

Both sides of the larynx are uniformly and symmetrically affected by the swelling, although in certain cases the inflammatory process seems to confine itself largely to one side of the larynx. Where the process goes on to the formation of an abscess this may be bilateral, but as a rule is unilateral.

Symptomatology.

At the onset of the attack there are usually chilly sensations, in rare cases a marked chill followed by slight fever, 100° to 100.5° perhaps, and then almost immediately by dyspnœa, showing the rapidity of development of the local affection. Impairment of voice, or aphonia, and stridulous breathing with both inspiration and expiration next occur, increasing to labored respiration and causing a florid and cyanotic face, the sufferings of the patient being shown by restlessness and an anxious expression. These symptoms of dyspnœa develop in from twelve to twenty-four hours, or on the third or fourth day, and unless tracheotomy or some other measure prevent, will result fatally.

In less severe cases the dyspnœic symptoms progress rapidly for

about twenty-four to thirty-six hours, when resolution occurs or else an abscess is formed, and its discharge ends the attack.

There is a still milder form of the disease in which a small abscess is formed on one side only of the larynx. Dyspnœa is not apt to occur in these cases, but the voice is impaired and there is a sense of fulness or pain in the neighborhood of the larynx; in from two to four days the disease has run its course. Pain on swallowing, some sensitiveness to pressure or movement of the larynx, and cough (in children, not adults) may be present.

Diagnosis.

The clinical history of the case will aid us in determining whether we are dealing with an exudation, a phlegmon, a passive œdema, or a foreign body, but examination by means of the laryngeal mirror, and possibly digital exploration of the larynx, must be our chief reliance. The index finger inserted into the fauces may easily reach the rounded, partially resistant masses distinctive of phlegmonous infiltration of the larynx, but our main dependence must be the laryngoscope, by means of which we shall find the laryngeal mucous membrane bright red in color, tense, somewhat glassy, and semi-opaque—in fact, in an inflammatory condition. The ventricular bands and the posterior face of the epiglottis will in severer cases show three large, rounded masses, obscuring the parts below, and leaving a contracted triangular opening for respiration. There is evidence of active inflammation but less tumefaction in milder cases; while a unilateral phlegmon will be recognized by the same general appearances (Fig. 54).

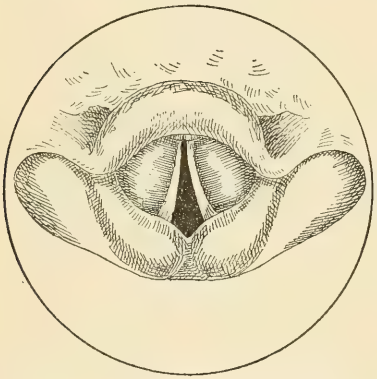


Fig. 54.—Acute Plegmonous Laryngitis.

Prognosis.

By the third or fourth day the process is usually at an end, although in cases severe enough to necessitate a tracheotomy the dyspnœic symptoms as a rule occur in from twelve to thirty-six hours after the onset of the disease. The breathing space is encroached upon by serous infiltration. The milder and more protracted cases terminate in the discharge of pus, from which we conclude that if at the end of thirty-six hours the development of grave laryngeal steno-

sis has not necessitated tracheotomy, the case will probably undergo suppuration without need for recourse to surgery.

Suffocation is the chief and practically the only danger of the disease.

Treatment.

We must attempt to secure local depletion by free scarification of the inflamed membrane on the face of the epiglottis and the swollen ventricular band, for which purpose we may use Tobold's concealed lancet or any other available laryngeal knife (Fig. 55), or even an ordinary curved bistoury, the blade of which must be wrapped with thread to within a quarter of an inch of the point. The laryngeal mirror will assist, or the finger may be passed into the fauces if there is not much dyspnœa. Scarification should be performed two or three times a day, to relieve the swollen blood-vessels, and allow the escape of serum.

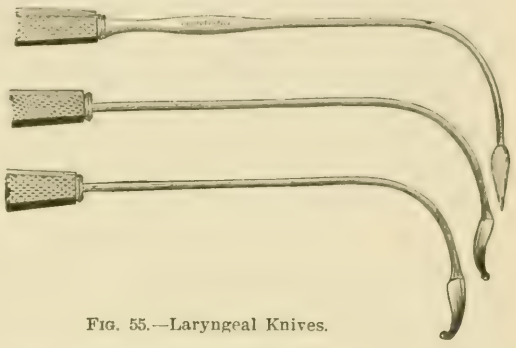


Fig. 55.—Laryngeal Knives.

Comfort may be derived from steam inhalations, but they are not curative. Blisters to the neck are useless, but leeches applied over the crico-thyroid membrane are good, while the persistent application of ice-bags or Leiter's coil is beneficial, and pieces of ice may be swallowed. A saline cathartic should be administered.

The necessary instruments for tracheotomy should be at hand, for the failure of remedial measures to arrest the development of dyspnœa for any length of time will necessitate the operation.

McEwen by the insertion of a common urethral catheter carried a case of phlegmonous laryngitis successfully through the stage of dyspnœa, but the insertion or retention of an intubation tube would be rendered almost impossible by the distortion of the larynx.

Tracheotomy is usually to be preferred to the higher operation, except in extreme cases where rapidity is the chief desideratum, because the opening should be as far as possible from the seat of inflammation. If pus can be reached by the knife it should be evacuated, but unless the operator is skilful, tracheotomy is a better resource.

Suffocation from severe dyspnœa occurring suddenly during the

physician's absence has so often occurred that no risks should be taken. A tracheotomy tube inserted before the occurrence of severe dyspnœa may save life and will relieve suffering.

Œdema of the Larynx.

I think it more accurate to reserve the term "œdema of the larynx" for that condition of the laryngeal cavity in which œdema is the chief and practically the only local lesion, and consists of an extensive serous infiltration of the soft parts. The essential lesion in phlegmonous laryngitis, perichondritis, and other inflammatory affections being inflammation, the term "œdema" as applied to them is misleading, although a certain amount of œdema is present. In syphilis, tuberculosis, and sometimes in malignant disease of the larynx an œdematous infiltration is met with, which in these cases also is merely a secondary development.

Etiology.

For the cause of the disease we must look to some condition, either general or local, outside of the larynx, which is capable of inducing a dropsical effusion in any other portion of the body. Renal disease is the most frequent source of this affection.

It is probable that a relaxed condition of the mucosa of the larynx will be more likely to favor the occurrence of local œdema than a normal state of that membrane. An aneurysm or other tumor in the cervical or upper thoracic region will by interference with the return of the venous circulation be apt to cause serous effusion into the larynx.

Under the name of "angioneurotic œdema of the larynx" has been reported a disease which is probably a vasomotor paresis resulting from general neurosis, and akin to the "acute circumscribed œdema of the skin" described by Quincke, which consists in the sudden appearance of certain defined areas of œdema which persist for a few days and then disappear only to be found upon some other portion of the body. The affection is hereditary. Placental degeneration has been known to cause œdema of the larynx in the newly born.

Pathology.

The essential lesion of this disease consists in the escape of liquor sanguinis from the blood-vessels into the submucous tissues of the larynx, causing swelling and distention, especially where there is much areolar tissue. In rare cases the œdema is confined to the ventricular bands without invading the epiglottis, but, as a rule, there are

three distinct serous sacs: one anteriorly, which starts on the posterior surface of the epiglottis, ascends to the epiglottic crest and may extend as far as the glosso-epiglottic fossæ; and two laterally, formed by distention of the aryepiglottic folds, extending to and involving the ventricular bands, invading the investing membrane of the arytenoid cartilages and the commissure.

The true cords and parts beneath are rarely involved. The whole pathological process consists of serous exudation, without further change in the exuded serum or in the tissues.

Symptomatology.

The attack usually occurs so suddenly and unexpectedly that the symptoms attending interference with the respiration are the first to be noticed. Loss of voice does not always occur immediately, but from extension of the œdematous swelling the movements of the glottis will be hampered and the vibrations of the cord interfered with.

Dyspnœa, especially upon inspiration, is the chief symptom; a noisy, stridulous sound is caused by the tendency of the swollen masses above the larynx to roll in when the current of air is inhaled and close the orifice in a valve-like manner. Pain is not a constant symptom, but there is a sensation of fulness and distention in the throat, and there is apt to be some difficulty and pain during deglutition.

So rapid is the progress of the swelling that in a few hours from the onset, or at latest in from twenty-four to thirty-six hours, extreme symptoms of dyspnœa may occur. Cyanosis, restless movements, an anxious expression of face, etc., will follow as the stenosis develops.

Diagnosis.

Our chief concern will be to decide upon the special form of laryngeal affection that is causing the stenosis which is usually unmistakably present, and here, as before, the laryngoscope or the examining finger will render assistance. Swelling of the epiglottis will be easily perceived in a digital examination as a large, round, semi-resistant mass beyond the base of the tongue. If the œdema be confined to the aryepiglottic folds, and if dyspnœa exist, the passage of the index finger beyond the epiglottis would not be well tolerated, in which case the laryngoscope should be used, as being alone capable of establishing a differential diagnosis between pure œdema, perichondritis, the presence of a foreign body, or other possible causes of the stenosis.

The field of vision would present the mucosa of the larynx with two or three large, rounded, swollen masses (Fig. 56) narrowing the lumen of the larynx and showing the small triangular opening which permits breathing to occur. The tense, semi-glassy, grayish, partly-transparent aspect of the membrane somewhat resembles a mucous polyp. In simple œdema there is an absence of the bright-red color of the parts seen in phlegmonous laryngitis. The possibility of angioneurotic œdema manifested in the larynx should be borne in mind, but cutaneous œdema would have preceded it. No other laryngeal lesions need be confounded with simple œdema.

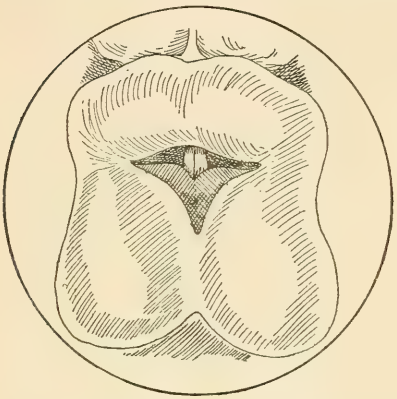


Fig. 56.—Œdema of the Larynx.

Prognosis.

The cases are rare indeed in which a favorable change in the disease occasioning the laryngeal œdema causes an arrest in the progress of this swelling. Therefore, unless remedial measures promptly and efficiently applied succeed in checking it, we may expect a dangerous dyspnœa to ensue within a few hours.

Treatment.

This should be addressed to the cause of the œdema, if it can be ascertained. One minim of the fluid extract of digitalis or the tincture of strophanthus should be given hypodermatically in case of deficient heart action. For kidney disease or cirrhosis of the liver, catharsis by means of half a grain of elaterium or the more rapidly acting drop of croton oil, and diaphoresis by one-eight of a grain of pilocarpine administered hypodermatically should be produced. The pilocarpine may be repeated in three or four hours, but its effects should be closely watched as it is a heart depressant and liable to cause œdema of the lungs. Alcoholic stimulants will counteract its action. A moist atmosphere must be maintained by boiling water over an alcohol lamp. The room should be warm. Wherever tumefaction is found in the larynx, free scarification as described under phlegmonous laryngitis should be practised, and will afford prompt relief from the pouring out of serum and subsidence of the swelling. The process may have to be repeated at the end of two or three hours.

As regards catheterization, intubation, and tracheotomy, the same rules apply that have been given under phlegmonous laryngitis.

Œdema as a secondary process, in tubercular and syphilitic disease, and angioneurotic œdema rarely become so severe as to necessitate local interference. When they do, however, they should be managed in the same way as an œdema resulting from disease of the heart or kidneys.

Croupous Laryngitis.

Believing croup and diphtheria to be separate and distinct, we give the name "croupous laryngitis" to a class of cases in which there is fibrinous exudation in the larynx, unattended by any of the septic features of diphtheria.

The affection is rare as compared with diphtheria, but from the careful study of many cases I am convinced that it does exist.

As all acute dyspnœic affections used to be called croup, and as the tendency of late has been to call them all diphtheria, the literature upon the subject cannot be relied upon to any great extent, and our discussion will be based chiefly upon personal observation.

Etiology.

I believe as firmly in the existence of a specific germ which causes croupous laryngitis, although it has not yet been isolated, as I do in the germ of diphtheria. The lymphatic tissues in the fauces, in which the primary deposit usually occurs, afford a convenient lodgment for disease germs and a favorable nidus for their propagation. The local inflammation set up by their presence takes on the form of croupous inflammation. In every case which has come under my observation exudation in the fauces preceded involvement of the larynx.

I think that there is good ground for the belief that a croupous deposit in the fauces may cause a croupous laryngitis, and this despite the fact that acute follicular tonsillitis is of very frequent occurrence in the autumn and winter and is rarely followed by croupous laryngitis. The somewhat feeble germ causing acute follicular tonsillitis, whose action is limited to the production of an exudation in the crypts of the tonsils, in a more active state gives rise to a fibrinous exudation covering the face of the tonsil; while there is another germ related to the croup germ, but infinitely more active, which produces diphtheria.

So little vitality is ordinarily possessed by the croup germ that after causing an acute follicular tonsillitis its activity ceases as a rule. Sometimes, however, it lodges in the fauces and produces a diffuse membrane on the tonsils. In some cases there are no laryngeal complications.

The point to which I wish to call attention is that there is a certain danger of similar disease of the larynx resulting from any purely croupous and non-diphtheritic deposit in the fauces. The danger is remote in diffuse membranous exudation on the tonsil, and still more remote in the case of follicular disease, but it should not be ignored.

The susceptibility to this disease, which is an important feature, commences at the age of one year, increases for six or eight years and then diminishes, and finally disappears at puberty.

The disease probably does not occur epidemically, but is rather sporadic and endemic; nor is it more contagious than acute follicular tonsillitis.

As an inflamed membrane affords a better nidus for the cultivation of a disease germ than a normal membrane, disease of the upper air passages must be regarded as a predisposing cause of a croupous inflammation, although not directly related to it.

Pathology.

The usual inflammatory process occurs in this affection, differing from that of catarrhal inflammation in that a large amount of fibrin contained by the escaping serum passes through and coagulates upon the surface of the mucous membrane. Epithelial cells proliferate and are imprisoned by the fibrin, forming in this wise the false membrane.

It is probable that the specific germ penetrates the tissues and sets up a catarrhal inflammation, and that by entering the circulation or by producing ptomaïnes which do so, it causes a hyperinosis which gives rise to the fibrinous inflammation or exudation.

Symptomatology.

Chilly sensations or a chill followed by a fever which is sthenic from its onset, and which may range from 102° to 104° F. on the first day, constitute the first symptoms of the disease. A hot, flushed skin and a rapid, bounding pulse accompany the fever, as do pains in the bones, restlessness, scanty high-colored urine, and a distaste for food, the latter being partly due to the dry, stiff, sore feeling in the throat and the extremely painful deglutition. The temperature remains practically the same for one or two days, and usually on the first, or perhaps not until the second or third day, the voice will become hoarse and metallic and will finally be reduced to a hoarse whisper.

Dyspnœa, more marked upon inspiration than expiration, with subclavicular depression, cyanosis, a pinched, anxious countenance, dilatation of the alæ of the nose, etc., are evidences of a membranous

deposit in the larynx and are usually marked by a rise of one or two degrees in temperature. Unless remedial measures arrest the disease or cause exfoliation of the membrane, the symptoms will increase in severity and death will occur from the third to the sixth day.

The relation, already alluded to, between this disease and diphtheria is still further shown by the presence of albuminuria in a certain number of cases, but as albumin is present in the urine in most acute infectious diseases it is not of special diagnostic value.

Diagnosis.

The important point consists in the differential diagnosis between croupous and diphtheritic inflammation. In the latter disease the false membrane is thick, yellow, and efflorescent and adheres so closely to the parts beneath that it cannot be separated from them without tearing the blood-vessels; at the end of twenty-four hours the exudation will show evidence of necrosis.

The croupous membrane is easily detached (which is the chief diagnostic point), and is a thin, clear, bluish-white deposit.

In very young children a laryngeal examination is rarely possible, but this is less of a disadvantage inasmuch as the fibrinous exudation in the larynx in diphtheria is practically a croupous membrane. Inspection of the fauces will have to be our dependence for the diagnosis, and should be sufficient, since I seriously question whether a case of laryngo-tracheal diphtheria, so called, ever occurs without a diphtheritic inflammation in the fauces. If at the end of twenty-four to thirty-six hours a faucial exudation has shown no localized tissue necrosis, the case must be regarded as a croupous disease.

Prognosis.

Death occurs from suffocation in croupous laryngitis, not from septicæmia nor from the severity of the fever. Were the therapeutic resources at our command able to overcome the pseudo-membranous deposit in the air passages or prevent its tendency to spread with rapidity into the trachea and bronchi, many lives would be saved. Opening the air passages is seldom successful when the trachea and bronchi have been invaded, but we are usually driven to it.

Treatment.

Mercury gives more hope of controlling the exudation than any other drug, although it has no effect upon the blood poison. A child five years of age should receive calomel or hydrargyrum cum creta suspended in milk or other fluid in full doses every two hours for the

first twenty-four hours, or until the stools are green and soft, when it may be given less frequently.

Tincture of iron comes next in value, and should be given in 1:8 of glycerin, a half-teaspoonful of which may be taken every two hours by a child of five years. Its action is both local and systemic; I believe the drug has a peculiar controlling influence over the blood condition spoken of as hyperinosis. Iron and mercury may be combined, but I think a better action is obtained by giving them alternately.

Locally, we may saturate the membrane with liquor ferri persulphatis by means of cotton wrapped on a probe, the application being repeated every two or four hours. This method is available for a faucial membrane, but where the process occurs in the larynx it is practically out of reach, and we shall have to rely upon the inhalation of steam, in the hope that we may cause rapid exfoliation of the membrane before asphyxia occurs. Lime inhalations are of great value, and may be repeated every four to six hours, simple steam inhalations generated by a spirit lamp and given under a tent made with sheet and blankets being constantly used during the intervals. I have never found any benefit from the use of the various drugs recommended, such as lactic aid, creosote, etc.

Emetics are less used than formerly, because it is considered that they do harm in a disease of so asthenic a nature as diphtheria, and the diphtheritic character of the large proportion of these cases has been recognized.

In croupous exudation they are undoubtedly of value, chiefly, however, in causing detachment and expulsion of the membrane during the act of vomiting. But as the membrane is not likely to be detached unless it has already begun to loosen, judgment must be exercised and the emetic administered at the time when the exudate would seem to be loosening. The yellow subsulphate of mercury or turpeth mineral is the best emetic for the purpose, and a dose should be given of not less than ten grains to a child five years old.

A somewhat powerful remedy and one to be used with caution is pilocarpine, which increases diaphoresis, stimulates the mucous secretions, and would seem to promote the separation of the false membrane. A twentieth of a grain may safely be given to a child five years old, and repeated at the end of from six to twelve hours under careful supervision.

Surgical interference will have to be resorted to in the majority of cases, and should be employed in time to prevent the long continuation of laryngeal stenosis, which depresses the vitality and produces morbid conditions in the mucous membrane. The O'Dwyer tube is

to be preferred to tracheotomy, and is to be inserted as soon as symptoms of dyspnœa become at all continuous; but if it fail to give relief, or cannot be retained in position, tracheotomy will have to be performed.

Perichondritis of the Laryngeal Cartilages.

A primary chondritis, so far as I know, is never met with, the pathological changes which occur in the laryngeal cartilages resulting usually from a morbid process having its origin in the perichondrium. Ossification, fibroid degeneration or chronic inflammation, and acute inflammation are the three varieties of morbid action in these tissues with which we are clinically acquainted.

Osseous degeneration, which is a change due to advanced age, often interferes with operations upon the larynx involving section of the cartilages, but otherwise has no clinical importance. Fibroid degeneration, which is of rare occurrence, is likewise an accompaniment of age, although it may occur in early life. It is usually caused by a strumous diathesis, and differs from the acute form of the disease only in the chronic course which it runs. Many cases of chronic subglottic laryngitis are probably complicated with this form of perichondrial inflammation.

Acute perichondritis is the form of the disease which we shall here consider, because of its more frequent occurrence than the other varieties, and because of the interest attached to the suddenness of its invasion, the gravity of the symptoms caused, the great deformity to which the laryngeal cavity is subjected, and the obscurity and difficulty of diagnosis which are often met with by the surgeon at a moment when prompt action is of importance.

Etiology.

The majority of cases occur as a result of exposure to cold. Among the exciting causes are typhoid fever, diphtheria, pneumonia, erysipelas, traumatism, and syphilis, the course in the latter being the same as in other cases, although the indications for treatment are different. Out of 33 cases, 2 of which occurred in my own practice, 9 were idiopathic, 9 due to syphilis, 11 followed typhoid fever, 1 diphtheria, 1 was due to traumatism, and 2 were probably due to a lordosis of the cervical vertebræ causing pressure upon the cricoid cartilage. Excessive use of the voice has been known to cause the disease, and pressure upon the cricoid cartilage by cervical vertebræ displaced in consequence of senile changes is supposed to be capable of inducing inflammatory changes.

Men are more liable to the disease than women.

Pathology.

An increase in vascularity, an augmentation of cell production, and the other usual inflammatory changes are the chief alterations undergone by the tissues. The cartilage itself becomes involved in the process, adding to the marked tumefaction. Pus may form and by burrowing separate the perichondrium from the cartilages and lead to their necrosis, or organization of the new cells may result in a true hypertrophic process, constituting properly a chronic perichondritis.

Usually only one, but sometimes all of the laryngeal cartilages are involved. Where the arytenoid is attacked, the disease frequently extends to the cricoid. If we include cases secondary to tubercular disease, Schrötter's classification of the cartilages in the order of frequency attacked is correct, viz., arytenoids, epiglottis, cricoid, and thyroid.

Symptomatology.

General malaise, chilly sensations, and, rarely, a marked chill usher in the attack, and are followed by some fever (100° to 101° F.), pain in the bones, headache, and anorexia. According to the cartilages involved there is more or less local impairment of function, consisting of interference with respiration, phonation, and deglutition. Fulness or distention of the parts and tenderness on pressure are the cause of much discomfort, but pain is not a prominent feature except where the disease is due to syphilis.

The Cricoid.—When this cartilage is attacked the inner perichondrium is always involved, causing a swelling which greatly encroaches upon the breathing space, the ensuing dyspnœa consequently becoming the chief symptom and an early one. Inspiration and expiration are almost equally affected. When the posterior surface of the cricoid is implicated, the pressure of food upon the inflamed tissues will cause dysphagia. Owing to the extension of inflammation to the mucosa of the lower part of the larynx, but chiefly to an infiltration of the muscular tissue which interferes with the motion of the cords, the voice becomes lost or is reduced to a mere whisper. The crico-arytenoidei laterales muscles are more actively attacked than the posticus.

Cough is not often a prominent symptom, although it may occur from the accumulation of mucus in the larynx. The symptoms usually persist with little change, until either suppuration or hypertrophic changes occur. I know of no case of spontaneous resolution, but it is theoretically possible.

The formation of a sequestrum from necrosis of the cartilage is

the final result of the process, and until it is thrown off, which may not be for months or even years, or removed, it will cause a production of pus which is discharged through fistulæ externally or into the larynx.

The Arytenoid.—Disease of this cartilage will give rise to a unilateral tumor which by encroachment upon the œsophagus will cause dysphagia, and upon the laryngeal cavity a certain amount of dyspnœa.

An effusion into the crico-arytenoid joint occurs in this form of the disease, practically causing an ankylosis and preventing all motion of the cord on that side, with the result that the voice becomes hoarse and low. Necrosis follows, but the final exfoliation takes place more rapidly than in the cricoid disease.

The Thyroid.—Either face of the cartilage and one or both wings may be affected, but as a rule the inner face only is the seat of the disease, which is moreover unilateral. The chief symptoms when the inner face of the cartilage is involved are impairment of the voice, due to involvement of the laryngeal mucosa and to swelling of the perichondrium projecting into the cavity near the ventricular band, and dyspnœa caused by the tumefaction from the extensive area of inflamed tissue. Where both wings of the cartilage are involved this interference with breathing becomes a very serious symptom. When the disease is unilateral the voice is hoarse and low, when bilateral it is practically destroyed. Disease of the outer surface causes a deformity externally, perceptible to the sight and appreciated by the touch, giving tenderness on pressure and sometimes localized pain.

Both wings and both faces of the thyroid cartilage are attacked only where all the laryngeal cartilages are diseased, when extensive necrosis of the part and death of the patient usually follow.

As a rule, an attack of thyroid perichondritis consists in the unilateral development of a localized inflammatory process in the perichondrium of one face of the cartilage, usually the inner. Suppuration occurs, followed by the formation of a fistula, usually near the median line, through which the pus is discharged either into the laryngeal cavity or anteriorly through the cutaneous tissues. The swelling then usually becomes less, and the symptoms show a trifling improvement.

The cartilaginous necrosis in thyroid perichondritis is apt to be more superficial and less extensive than in disease of the cricoid or arytenoid, owing to the more abundant supply of blood-vessels. The affected portions disintegrate and are discharged, and in the course of a few months or a year the parts heal without great loss of tissue or injury to phonation.

The Epiglottis.—This is never affected in a similar manner as the

parts just described, owing to its fibro-cartilaginous structure. Perichondritis in this situation is usually secondary to tuberculosis, syphilis, carcinoma, etc., and results in ulcerative action instead of in abscess and necrosis.

Diagnosis.

That we have to deal with acute inflammatory disease is shown by the suddenness of the onset and the fever. Perichondritis, croupous exudation, and acute submucous laryngitis being the only acute inflammatory diseases causing dyspnœa, we should expect the diagnosis to be simple, yet experience shows that such is not the case. The chilly sensations at the onset of a croupous laryngitis and the fever are both more marked, the temperature ranging from 102.5° to 104°. If no croupous exudation is seen in the fauces, it should be revealed by the laryngoscopic mirror. In submucous laryngitis there is a conspicuous, symmetrical, bilateral swelling of an inflammatory nature of the mucosa of the larynx, while in perichondritis the tumefaction, while presenting the aspect of acute inflammation extending to neighboring parts, is usually unilateral, irregular, and asymmetrical.

The physician is usually summoned in an emergency, when it is not possible to ascertain whether there may not have been some pre-existing obscure growth or malignant tumor. Hence the diagnosis will have to be based on the exclusion of other acute affections and the nature of the febrile movement, together with the use of the laryngeal mirror. Affection of the cricoid will cause a perceptible tumefaction of irregular outline and nodular character, which projects into the larynx below the glottis, and infringes upon the breathing space (Fig. 57). The tumor is below the cords, but appears to project into the supraglottic larynx, and by pushing up the arytenoid and vocal cord brings the latter against the ventricular band and more or less obliterates the ventricle. The movements of the larynx on the affected side are interfered with or prevented.

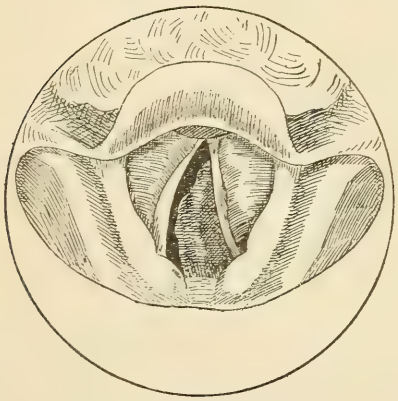


Fig. 57.—Laryngoscopic Image in Perichondritis of the Cricoid Cartilage.

Careful study of the parts by means of the laryngeal mirror enables one to outline the swelling originating on the posterior segment of the cricoid, its usual starting-place.

Extensive tumefaction is also found in disease of the arytenoid cartilages, which are covered by the swelling. Idiopathic perichondritis is almost always unilateral, and the diffuse bright-red color shows it to be purely inflammatory, while tuberculosis, the only condition liable to be confounded with it, is usually bilateral and exhibits minute, scattered whitish-gray points on a pale gray-pink membrane.

Palpation and inspection, and the careful study of symptoms, will show whether the thyroid is affected externally. Involvement of the inner face will be seen by the laryngeal mirror to cause a distinct swelling, which so pushes forward into the laryngeal cavity near the ventricular band as to narrow the breathing-space and practically hide the true cord. Surrounding tissues are also affected by the acute inflammatory process; the presence of this inflammatory process will serve to exclude all neoplasms.

Prognosis.

The course of these cases is a somewhat prolonged one, but not necessarily fatal except from laryngeal stenosis, which can be averted by means of a tracheal tube. The rare cases in which all the laryngeal cartilages are involved, and which are usually secondary to tuberculosis, carcinoma, or some systemic disease, almost invariably have a fatal result.

In cases of cricoid perichondritis there is great danger of permanent respiratory obstruction, as we may see by the fact that of seventy cases compiled by Lüning which survived tracheotomy sixty were obliged permanently to wear a tracheal tube. These cases, however, all followed typhoid fever, which in some way predisposes to persistent laryngeal stricture. In thyroid perichondritis the sequestrum formed is apt to disintegrate and be discharged, or else to become so large that an operation is imperative, wherefore the course is usually more rapid than in disease of the cricoid. Pus may burrow beneath the deep tissues of the neck into the anterior mediastinum, causing suppurative inflammation, but this danger is remote.

Perichondritis of the arytenoid causes a destruction of the cartilage, which is usually expelled, and results in a permanent ankylosis of the crico-arytenoid joint, with some injurious effect upon the voice. There is also danger of extension of the disease to the cricoid.

Treatment.

In the acute stage active general and local antiphlogistic measures must be used. The ice pack or Leiter's coil should be applied externally, while pellets of ice are given internally and local blood-let-

ting is obtained by means of leeches or wet-cupping. Absorbents, as mercurial ointments, iodoform, or iodol ointments may be applied externally, or a solution of iodine and glycerin may be applied to the laryngeal mucosa. Magendie's solution of morphine, or a ten-per-cent. solution of cocaine may be sprayed in the laryngeal cavity in the case of great pain. Blue mass or calomel, followed by a mild saline cathartic, must be used to keep the bowels freely open. Iodide of potassium should always, even when there is no specific history, be administered at the beginning of the acute stage, and be kept up after the acute symptoms have passed away.

Treatment for relief of the dyspnœa, tracheotomy when called for, management of the sequestrum, and relief of stenosis may all be demanded at a later stage. Laryngotomy is better than tracheotomy, because by it we obtain some local blood-letting and an opening through which we can reach the sequestrum in case of cricoid perichondritis, or accumulations of pus in thyroid perichondritis.

The presence of a sequestrum stimulates connective-tissue formation, and therefore is liable to cause permanent deformity and stenosis. The dead portion should be removed as soon as it becomes detached from the diseased perichondrium. This can be done through the tracheal opening, but it would be safer to enlarge the opening in either a vertical or a lateral direction, and still better to reinsert the tube lower down.

The use of the Luer valve in connection with the tracheal tube is, to my mind, of great importance for the conservation of the voice, and restoration of the breathing space, especially where there is ankylosis of the crico-arytenoid joint, for the forcible expiration caused by this valve occasions not a little motion.

In some cases it will be necessary to cut laryngeal adhesions, or to dilate the strictures by bougies. Heine's case, in which a large portion of the larynx was removed and an artificial larynx inserted, will show the extreme measures which may have to be resorted to, laryngeal strictures calling for the utmost skill of which the physician is possessed.

Laryngeal Hemorrhage.

Excluding those cases in which hemorrhage is due to syphilitic or tubercular ulcer, malignant disease, the presence of a foreign body, etc., we apply the term to cases in which ruptures of the blood-vessels of the larynx cause hæmatomata by escape of blood into the mucous tissues, or hæmoptysis by its penetration through the surface of the mucous membrane. The introduction of the laryngoscope led to a

recognition of the fact that the laryngeal mucous membrane was frequently a source of hemorrhage, and modified the then prevalent opinion that all cases of hæmoptysis were a result of pulmonary disease.

Etiology.

The majority of writers at the present day appear to hold the opinion that inflammation of the laryngeal mucous membrane forms an essential part of laryngeal hemorrhage, as is evidenced by the frequent use of the term laryngitis hemorrhagica. It seems to me that the frequency of catarrhal laryngitis and the great rarity of hemorrhage of the larynx constrain us to the view that simple inflammation is not even an actively predisposing cause of the attack. Laryngeal hemorrhage I consider far the better term, as defining the chief symptom of the disease without misleading us as to its origin.

The result of studying reported cases would seem to be that general conditions are as potent factors in the causation of the disease as are local lesions. Among them we note the hemorrhagic diathesis, cirrhosis of the liver, and dilatation of the heart, the general debility of phthisis, anæmia, and malnutrition; one case was due to vicarious menstruation, and one to pregnancy. In a few cases the patient is apparently in perfect health, and the cause of the bleeding is hard to discover. It may lie in some local condition which weakens the walls of the blood-vessels of the larynx, such as slight erosions following a chronic laryngitis. The anterior face of the arytenoids is one of the few localities of the upper air tract where erosions occur, and the escape of blood has in many cases been traced to this point. Straining of the voice by excessive use is an exciting cause of the attack, as are coughing, vomiting, straining, violent exercise, and anything causing a superficial plethora. An acute laryngitis has been known to occasion an attack.

Pathology.

We may have an extravasation of blood beneath the surface, or an hæmoptysis caused by rupture of blood-vessels on the surface of the mucosa. It usually occurs over a limited area or at a single point. It is rather a diapedesis than the result of predisposing local causes, and therefore may arise from any part of the laryngeal cavity, although its usual seat is either the ventricular bands or the true cords. When caused by erosion of the mucous membrane, it occurs principally at the insertion of the cords posteriorly or on the anterior face of the arytenoid cartilage. According to Brown-Séquard, vasomotor disturbances of the mucous membrane may constitute a promi-

nent predisposing pathological condition. The disease scarcely ever proving fatal, there is seldom an opportunity for careful study of the local condition. In one case, however, the patient having died of some concurrent disease, the parts were minutely examined and found to be in a state of vascular plethora with an extravasation of red blood corpuscles, the diapedesis being due to cirrhosis of the liver and dilatation of the heart.

Symptomatology.

Laryngeal irritation and a tendency to cough, with alteration or loss of voice are the symptoms of simple extravasation or concealed hemorrhage. Dyspnœa will occur if the hæmatoma be large enough to infringe upon the breathing-space.

Hæmoptysis is of course the most prominent symptom of an open hemorrhage. With a slight effort at clearing the throat the blood comes up easily in small masses unmixed with saliva, this serving to distinguish it from pulmonary hemorrhage which it resembles in color. When small in amount it may appear as dark-colored clots, from having lodged in the larynx. It may continue for days with but little change.

Profuse hemorrhage is rare, and dyspnœa is seldom present, except when a clot lodges in the larynx and interferes with respiration.

Diagnosis.

In hæmoptysis the blood may come from the lungs, the larynx, the nose, or the naso-pharynx, in the latter case lodging in the pharynx or larynx and being subsequently expectorated; the upper air passages should therefore be included in a careful examination. In pulmonary hemorrhage the trachea is found coated with blood as far down as the bifurcation, the blood is usually admixed with mucus, and the lungs themselves will yield evidence of disease. In laryngeal hemorrhage the blood is only with extreme rarity found in the trachea; it may be expectorated in inspissated or dry clots, and examination of the mucosa of the larynx should discover the immediate source of the hemorrhage.

The laryngoscope, in cases of concealed hemorrhage, will clear up the diagnosis by showing a rounded, deep red or purplish tumor projecting from one of the walls of the larynx, so decidedly a hæmatoma in appearance that it cannot be confounded with a neoplasm or with œdema of the glottis.

Course and Prognosis.

I do not know of any case of fatal hemorrhage of the larynx, but an attack often persists in its course for days and weeks, which is

natural enough if it is the result of a general blood condition. There is no need for alarm in its long continuance, as it is not the precursor of severe hemorrhage, nor does it involve any danger of a pulmonary hemorrhage ensuing, even where it is due to the hemorrhagic diathesis, cirrhosis of the liver, or heart disease.

Treatment.

In case of hæmatoma where the tumor is large enough to cause definite symptoms, it should be opened by incision and the clots evacuated, a procedure which can be done with little danger of further hemorrhage. Small masses may be allowed to remain and undergo absorption.

Open hemorrhage may usually be readily controlled by some simple astringent, as one of the following: Ferri et aluminis sulphas, gr. x. to the ounce; liquor ferri persulphatis, ℥ x. to the ounce; acidum tannicum, gr. xx. to the ounce; argenti nitras, gr. v. to the ounce. A laryngeal atomizer should be used to apply the solution once or twice a day according to indications.

The patient should avoid exercise, the voice must have absolute rest, and the food should be bland and unirritating. No warm or highly seasoned foods or drinks are to be taken. Bits of ice held in the mouth will give relief, and may be applied externally to the neck.

Opiates must be given somewhat freely to subdue a cough or a tendency to clear the throat. Alcohol and tobacco are to be forbidden.

The action of remedies held to have a systemic effect on hemorrhage, such as ergot, iron, or sulphuric acid, is not to be depended upon, but they may be given if the amount of hemorrhage be severe. As malaria may temporarily aggravate the trouble, quinine should be given in full doses if the hæmoptysis shows signs of periodicity.

Syphilis of the Larynx.

Syphilis appears in the larynx as, 1st, the primary lesion; 2d, erythema; 3d, the mucous patch; 4th, the superficial ulcer; 5th, the gummy tumor; 6th, the deep ulcer; 7th, cicatricial stenosis.

The Primary Lesion.

Only one case, to my knowledge, has been reported. It occurred on the free border of the epiglottis on the left side, and in its local features seems to have differed in nowise from an ordinary buccal chancre.

Erythema of the Larynx.

This belongs to the secondary stage of the disease, and occurs usually from the fourth to the sixth month, although it has appeared as early as four weeks and as late as two years or more after the primary lesion. Pathologically it resembles cutaneous erythema but develops after the skin eruption has subsided. The invasion is insidious. There is no local pain or difficulty in swallowing; there is a certain amount of impairment of function, and if the cords are involved the voice may be affected or lost.

Pathology.

The essential lesion consists in a round-celled infiltration of the mucous membrane, occasioning some swelling of the tissue and markedly interfering with the return circulation of the blood, which causes a perceptible amount of venous turgescence. This infiltration may by an equable diffusion cause a uniform hyperæmia or, rarely, it may occasion an irregular vascular plethora, which gives a mottled appearance to the membrane. In acute catarrhal inflammation of the laryngeal membrane the first change consists of a vasomotor paresis, which is followed by a dilatation of the blood-vessels, and then by cellular infiltration. In syphilis the cellular infiltration comes first, and the vascular disturbances are secondary. The mucosa of the posterior surface of the epiglottis, the ventricular bands, and the ary-epiglottic folds is usually affected and occasionally, but rarely, the vocal cords are attacked. In a protoplasmic form of this disease described by Fournier the parts are more swollen, the subjective symptoms are more prominent, and localized prominences may sometimes be seen.

Diagnosis.

The peculiar, dusky, sombre, dark-red hue of the membrane in contrast to the scarlet color of an acute idiopathic inflammation constitutes our chief reliance in the diagnosis of syphilis by ocular inspection. The mottled appearance of the membrane mentioned above is still clearer evidence of a specific taint. There is diffuse venous turgescence in this form of the disease, but this is more intense in circumscribed areas which are from one to four lines in diameter. It should not be easy to confound the hyperplastic form with its characteristic color and extensive and uniformly diffused swelling with a simple inflammatory process of mucous membrane or perichondrium. The clinical history of the case and the success of specific treatment are our only means of recognition of a syphilitic erythema, with the exception of the methods given above.

Course and Prognosis.

This disease yields readily to appropriate treatment, but if neglected may remain for weeks and months at the same stage, without, however, developing any graver manifestation of syphilis.

The Mucous Patch.

This lesion also belongs to the secondary stage of syphilis, and is of extremely rare occurrence, so rare indeed that many writers doubt that it ever occurs at all. We believe it to have been too frequently reported by reliable observers to admit of a doubt.

It may make its appearance six weeks or twelve months after the primary invasion, and is most frequently situated on the upper surface of the vocal cords, appearing next in the order of frequency on the epiglottis, the arytenoids, and the ventricular bands. The symptoms depend upon its location. Affection of the cords occasions impairment of the voice, and a patch near the free border of the epiglottis will cause some slight pain in swallowing. It does not appear to set up secondary inflammatory changes.

The pathological lesion consists merely in a round-celled infiltration of the epithelial layer of the mucous membrane. Here, as elsewhere, it shows a tendency toward persistency and recurrence. The lesion may be single or multiple.

Diagnosis.

The mucous patch in the larynx is visible as a small, peculiar grayish patch slightly raised above the surface of the mucous membrane, which may present an areola of increased redness.

The Superficial Ulcer.

This lesion may develop from a mucous patch, or result from the breaking down of a superficial gummatous infiltration. It belongs to the secondary stage of syphilis, and occurs from two to seven years after the primary sore. It causes few symptoms, and the destruction of tissue is not rapid. Some impairment of voice may occur, also a secretion of muco-pus tinged with blood, but no cough, pain, or difficulty in swallowing.

The clinical history of this lesion being marked by frequent recurrences and exacerbations, it has been called "relapsing ulcerative laryngitis." I believe that there are relapses of a purely catarrhal type, but not syphilitic relapses with frequent deposits of gummatous material. A localized deposit occurs as a result of the syphilitic virus in the blood, with which deposit the activity of the virus for the

time ceases; the subsequent breaking down of the deposit into an ulcer is due to local causes, and is therefore practically a local process although originally caused by a specific blood poison. Recurrence of ulceration would necessarily be due to a new deposit of gummatous material, which does not as a rule occur in superficial ulcers. From a somewhat extensive personal experience in the study of diseases of the upper air tract, I hold that this view is true in the case of gummatous infiltration and ulceration in this situation, the process differing notably from that of cutaneous syphilis.

The chief features of a superficial syphilitic ulcer of the larynx (which is of infrequent occurrence) are its rounded, ovoid form, its bright-yellow color, and the absence of an areola of any importance. It is but slightly excavated, and secretes a yellow pus tinged with blood, containing perhaps a few shreds of necrotic connective tissue. Its characteristics are so marked when it is situated upon the ventricular bands, the epiglottis, in the glosso-epiglottic fossa (Fig. 58), or upon the posterior wall of the larynx that it should be easy of recognition; but on the vocal cords, where this form of ulcer is most frequently situated, a most careful inspection with a good illumination will be needed, for the membrane is so limited in extent that the tumor is naturally small, and there is but a slight accumulation of secretion. A solution of continuity and loss of tissue will be found, however.

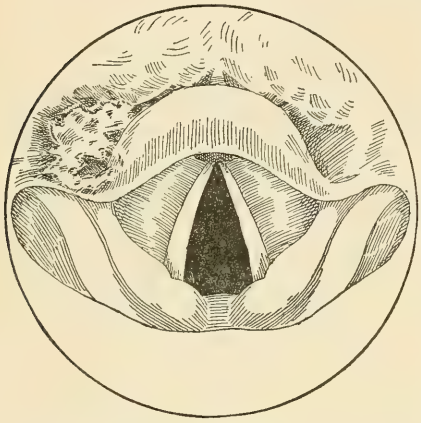


Fig. 58.—Superficial Ulceration in the Left Glosso-Epiglottic Fossa.

It should not be forgotten that syphilis may develop in connection with a tuberculous process.

The Gummy Tumor.

In constitutional syphilis the larynx is rarely attacked by the lesions belonging to the secondary stage, but as a rule remains unaffected until five or ten years or more after the primary sore, and is then the seat of a gummatous deposit involving the deeper tissues of the mucosa and periosteum, causing a gummy tumor and deep ulceration.

In the majority of cases a gummatous infiltration in this situation breaks down with such rapidity that the ulcerative process is fully

established by the time that the lesion is first seen, which is probably due to the fact that the larynx is the site of a constant functional activity. Sometimes, however, from some unknown cause, a delay occurs in the breaking down of the tissue, and the lesion may be seen as a single or multiple tumefaction or as a diffuse infiltration.

Symptomatology.

In a gummy tumor the deep layers of the mucous membrane are the seat of an infiltration, which by extension to the perichondrium may cause pain. The localized pain is less pronounced where the soft parts, such as the aryepiglottic folds or the ventricular bands, are involved, but some discomfort is caused by pressure during deglutition, which is also the case when the posterior walls of the larynx are affected. According as the motion and the shape of the vocal cords are modified will be the impairment of the voice. Dyspnœa may result from encroachment on the respiratory tract or from an œdema due to the presence of the growth.

Diagnosis.

A gummy tumor of the larynx appears rather suddenly, and unless softening and ulceration take place, undergoes no further change after its full development is attained. Whether single or multiple, it is found to be a smooth, symmetrical, rounded tumefaction, usually covered with healthy mucous membrane, which, however, may become inflated from other causes. In size it may vary from a pin head to a large cherry, and may occur in any part of the larynx (see Fig. 59). We should suspect syphilis wherever the clinical history of the case and the subjective symptoms point to it in connection with a rapidly developing tumor of smooth, rounded outline. In doubtful cases the effect of iodide of potassium administered internally will in a few days decide the question. Even when the history of primary or secondary syphilis is absent, and the case has the appearance of malignant disease, antisyphilitic remedies should be administered in order to eliminate completely all possibility of the growth being of specific character.

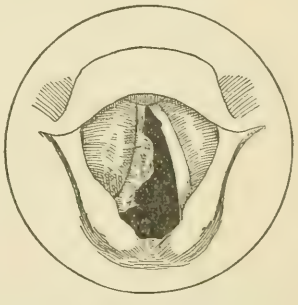


Fig. 59.—Gummy Tumor of the Left Vocal Cord.

Course and Prognosis.

The almost constant tendency of a gummy tumor to break down into an ulcerative process may sometimes be absent, or may be delayed for months or even years.

The Deep Ulcer.

This belongs to what is clinically known as the tertiary stage of the disease, seldom occurring under five years after the primary sore and usually being delayed until after ten years. It is caused by the breaking down of gummatous deposits in the deep portions of the mucosa proper. As we mentioned before, a deep-seated gummy deposit may remain without alteration for as long as two years even, but this is unusual, the rule being that of great rapidity of ulcerative action. The invasion of syphilis in the larynx is usually insidious, and the lesion may be present for some time without occasioning subjective symptoms at all in proportion to the severity of the local process. A little pain and tenderness on pressure may be present or absent at the time of the primary deposit. The voice is impaired or lost. Muco-pus admixed with shreds of black, necrotic tissue is abundantly secreted at the time of ulceration. Hemorrhage is rare. Dyspnœa may occur from encroachment of the tumor upon the air passages, but after ulceration has set in it is rarely a symptom unless œdema ensue, and then not to any threatening extent.

The epiglottis, vocal cords, ventricular bands, and the arytenoid commissure are the parts attacked by the ulcer in the order of frequency. When the soft parts are attacked the complications are limited to œdematous or vascular tumefaction, but near the cartilages, such as the arytenoids or cricoids, the infiltration often extends to the perichondrium, causing a perichondritis, which when of sufficient extent is followed by necrosis. One of the arytenoid cartilages is not infrequently destroyed in this manner, and a case is on record in which the necrosed arytenoid became detached, lodged in the glottis, and caused death by suffocation. Pain in swallowing, swelling of the membrane covering the cartilage, lessened motion of the cords, and perhaps dyspnœa are the symptoms indicating this complication.

In the event of perichondritis of the cricoid there is localized pain and tenderness on pressure, alteration of the voice, and above all dyspnœa, which comes on with rapidity and usually necessitates tracheotomy. After operation the course of the disease is similar to that of idiopathic perichondritis of the cricoid—namely, necrosis of the cartilage, and the formation of a sequestrum which remains embedded in the tissues of the neck for months or years, causing a discharge of pus into the larynx or through external sinuses on the neck. It is only in the late stages of laryngeal syphilis that perichondritis and necrosis of the thyroid cartilage occurs, and the symptoms which it occasions are masked by the graver lesions in other parts of the larynx.

The tertiary ulcer is very frequently found upon the epiglottis, but this being a fibro-cartilage, we have a process of caries and destruction of the organ by erosion instead of necrosis and the formation of a sequestrum (Fig. 60).

Perichondritis is not apt to occur as a complication with the first development of the tertiary ulcer, but later. After gummatous infiltration takes place the blood poison is for the time being latent, and the ulcer cicatrizes, causing contraction and some impairment of function, after which there may be several months of immunity. This is followed by a new outbreak, with deposit of gummatous material and its ulceration, and these "relapses" may occur through a period of ten or fifteen years, if the case is not treated or the blood poison not eliminated. Simple attacks of ulceration characterize the early years of the disorder, but later, when the larynx has been permanently injured, its lumen narrowed by cicatricial contraction, and its nutrition interfered with by the obstruction to circulation, the disease attacks deeper structures, and perichondritis and necrosis make their appearance.

FIG. 60.—Destruction of the Epiglottis from Syphilitic Ulceration.

Localized œdema is more frequent in the tertiary stage of syphilis than in any other, but I do not consider it of great importance or the cause of serious symptoms.

Diagnosis.

There should be little difficulty in recognizing the deep ulcer, with its sharp-cut edges, dark-red areola, excavated surface, and profuse secretion of pus admixed with necrotic tissue.

In tuberculous disease the areola is absent, the mucous membranes are markedly pale, the surface is whitish-gray, on a level with the membrane, there is no depression, and the ropy mucus secreted is small in amount.

Lupus is distinguished by an irregular contour and a slightly injected membrane, but there is no ulceration, no pus, no exfoliation of necrotic tissue.

The distinct tumor with possibly an eroded surface, but no distinct ulcerated surface with pus secretion, should serve to indicate the presence of sarcoma.

Carcinoma presents a hard, nodular tumor and a ragged ulceration without sharply cut edges; the crater-like aspect of the syphilitic ulcer and a well-marked areola are wanting. There is the tendency to hemorrhage, and moreover, in the ulcerative stage of carcinoma the cervical lymphatics are large and swollen, and cachexia is usually present.

Prognosis.

In its early stages laryngeal syphilis has a fair chance of being arrested in its course; still where a deep ulcer is found it is best to be guarded in giving too optimistic an opinion in regard to the success of treatment, and especially complete restoration of function. Tertiary syphilis of the larynx is evidence of an active blood poison, there is always destruction of tissue, and the cicatrization which attends healing necessarily leads to some deformity of the organ.

Cicatricial Stenosis.

I believe that an ulcerative process always precedes the laryngeal stenosis which occurs in the late stage of syphilis. After the localized necrosis which follows a gummatous deposit has sloughed away Nature, as always, endeavors to restore the parts by the ordinary process of repair. Syphilis especially causes vigorous and extensive cicatrization after ulcerative action. Cicatricial tissue after organization contracts vigorously, and as a consequence the outline of the larynx is in great measure destroyed, its lumen distorted, the respiratory passage much encroached upon, and the voice seriously affected.

Many observers seem to think that the lesion is caused by a sort of submucous laryngitis due to a deep-seated infiltration, which laryngitis in its later stage develops the stenosis without ulceration. In my experience there has never occurred a case in which I was not justified in considering the stenosis to be due to a contracting cicatrix following a tertiary ulcer. It is, therefore, my belief that a laryngeal stenosis not due to the presence of a gummy tumor or œdema points to the previous existence of an ulcerative process.

The voice is of course affected by the stenosis, and may be impaired or so completely lost that the patient is obliged to whisper; the tone in either case is coarse, harsh, and raucous and thoroughly characteristic of late laryngeal syphilis. Cough and increased secretion are an evidence of disturbance lower down in the air passages. Localized pain and tenderness on pressure accompany a new gummatous deposit, as do difficult and painful deglutition, although the latter may also be due to coincident infiltration of the pharynx or parts above. Dyspnœa is more marked upon inspiration because during

this act the softer tissues above the vocal cords roll in upon the glottis and increase the stenosis, but it also occurs during expiration. It is increased on exertion. At first not marked, the dyspnœa increases in intensity with the gradual narrowing of the larynx from the contraction of the cicatricial connective tissue.

Although in some cases a laryngeal stricture may develop after a single attack, the more usual course is that of a series of outbreaks of gummatous infiltration, followed by ulceration, with a gradual increase of the stenosis. The mucous membrane becomes the seat of a catarrhal inflammation, predisposing the patient to colds which develop as simply catarrhal laryngitis, or submucous laryngitis with œdema, with consequent marked aggravation of the symptoms. The more advanced the stage of the disease and the greater the extent of the stenosis, the more liable are these catarrhal attacks to occur, and the more apt to cause grave symptoms. Suffocation may ultimately ensue unless tracheotomy is resorted to.

Diagnosis.

The existence of a typical tertiary ulcer in connection with the stenosis would, with the aid of the laryngoscope, greatly simplify the diagnosis, but even where there is pure cicatricial contraction without ulceration an experienced eye will be able to find certain appearances characteristic of the ravages of syphilis. The cicatrices following the deep ulcer of syphilis give rise to a peculiar distortion of the organ, such as is caused by no other destructive ulcer. The ulcerative process in tuberculosis exhibits only feeble efforts at repair, and causes little cicatrization. But the reparative effort of nature in syphilis results in large bands of dense fascia-like cicatrices, which pull and distort the organ entirely out of shape. The mucous membrane is pale and bloodless in tuberculosis, while in syphilis the portion not included in cicatrization is congested, and of a dark purple hue. Carcinoma before ulceration is a distinct, clearly defined tumor encroaching upon the organ but not pulling or deforming it, and, moreover, it does not give rise to cicatricial bands.

Lupus is really the only disease which may render a diagnosis difficult, for it, just as syphilis of the larynx, causes an asymmetrical distortion of the organ and at times a peculiar venous hyperæmia of the mucous membrane. But lupus does not occasion true ulcerative action, and attempts at cicatrization are feeble and never result in the large bundles of cicatricial tissue caused by syphilis. The clinical history of the case, the absence of a history of primary and secondary lesions, and the recurrent outbreaks characterizing syphilis will help to clear up the diagnosis. The whole cavity of the larynx is so distorted

by the disease that it is difficult to ascertain the exact point of stricture. The ventricular bands are thickened and usually adhere to the vocal cords; the opening into the ventricles is obliterated. Especially anteriorly both true and false cords are more or less adherent, and careful examination of the parts below is rendered difficult by the drawing down of the epiglottis upon the larynx. It may indeed be almost or quite destroyed, in which case the entrance to the larynx is seen as a rounded, ragged circle, below which in a gradually narrowing space we perceive the ventricular bands merged into the true cords, covered in places by cicatricial tissue, and separated by puffy, purplish mucous membrane. A narrow breathing-space is usually left in front of the posterior commissure, as the adhesions between the cords generally occur anteriorly (see Fig. 61).

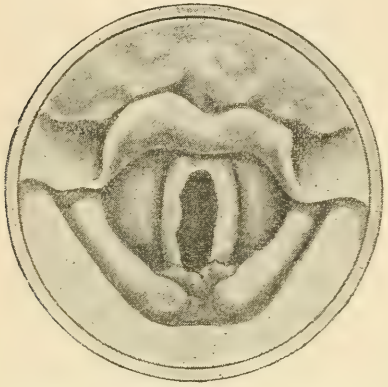


Fig. 61.—Cicatricial Stenosis of the Larynx the Result of Syphilitic Ulceration.

No two cases are exactly similar and we may have marked stenosis as the result of ulceration and cicatrization, and chiefly confined to one side of the larynx. The stenosis is materially increased by the usually early involvement of the crico-arytenoid articulation on one or both sides, resulting in a fixation at or near the median line, the situation of the fixation being probably due to the fact that the abductor muscles are weaker than the adductors.

Course and Prognosis of Laryngeal Syphilis.

Laryngeal syphilis with its sequelæ may extend over a number of years. If treated at the onset it may be arrested, but if ulceration have occurred, some tissue will necessarily be destroyed, and no local or general treatment can control the cicatrization and contraction which are sure to follow. Prognosis as to the question of vocal impairment and preservation of the air passage must be based upon the amount of destruction which has taken place at the time, and the probable amount of resulting contraction.

Dyspnœa and suffocation are the only elements of danger to life, and can be averted by the prompt performance of tracheotomy.

Treatment of Laryngeal Syphilis.

The Primary Ulcer.—Laryngeal chancre comes under the generally accepted rule that it is better to wait for secondary manifestations than to attempt to treat the primary sore.

Erythema.—General remedies will readily overcome this lesion. Topical applications such as were mentioned in the treatment of acute simple catarrhal laryngitis may be used if the local process assumes an aggravated form.

The Mucous Patch.—In the larynx it is of a less persistent and recurrent type, and has less tendency to spread than when situated on the soft palate and pharynx. Internal medication seems to overcome it, but if local treatment is needed cauterization should be applied.

The Superficial Ulcer.—Cleansing lotions and iodoform or europhen locally applied constitute the treatment for this lesion. A sixty-grain solution of nitrate of silver has been recommended, but I have obtained the best results from iodoform applied by a Lefferts powder blower.

The Deep Ulcer and Gummy Tumor.—Inhalations of corrosive sublimate, from 1:1,000 to 1:500 have been highly praised, and may be administered by means of an ordinary atomizer, or by the Globe inhaler in cases which do not respond promptly to measures already recommended. Our main reliance here must be upon the administration of large and increasing doses of iodide of potassium.

Cicatricial Stenosis.—Internal medication is of course useless in relieving a condition due to cicatricial contraction and inflammatory adhesion of the tissue. It should be known that iodide of potassium, even in moderate doses, may cause an attack of iodism, usually in the form of iodic coryza, but sometimes manifested as an iodic laryngitis, whereby the dyspnœic symptoms are often dangerously aggravated. Although, when the larynx was in a normal condition, I have never seen a case of acute laryngitis caused by potassium iodide, I have in several instances observed it in laryngeal syphilis, wherefore if this drug be administered, its effects should be carefully watched.

Stenosis of the larynx, not being amenable to internal medication, should be regarded as in reality a stricture of the air passages, to be treated in the same manner as stricture of the urethra, œsophagus, or any other tract, namely, by continuous dilatation, divulsion, or section.

Schrötter devised a series of metallic bougies somewhat triangular in shape, about one and a half inches long and one-quarter to two-thirds of an inch in diameter, there being twenty-four sizes. There is an eye in the upper end of the bougie to which a cord is fastened, while the lower end is expanded into a small knob. They were de-

signed for use in cases where tracheotomy had already been performed. The patient is prepared for their passage by the daily insertion of a probe into the larynx. A cord is fastened to the eye of the bougie passed through a bent cannula and attached at the distal extremity of a handle into which the cannula is inserted, as shown in Fig. 62. The bougie is passed into the larynx, and its lower end inserted through the fenestra of the tracheal tube where it is seized and held by a small pair of spring forceps inserted through the cervical end of the cannula. The bent cannula which is used for inserting the bougie is now withdrawn, leaving the cord protruding from the mouth; this is now secured by tying it round the neck, and the bougie is left in place for a length of time varying with the tolerance of the

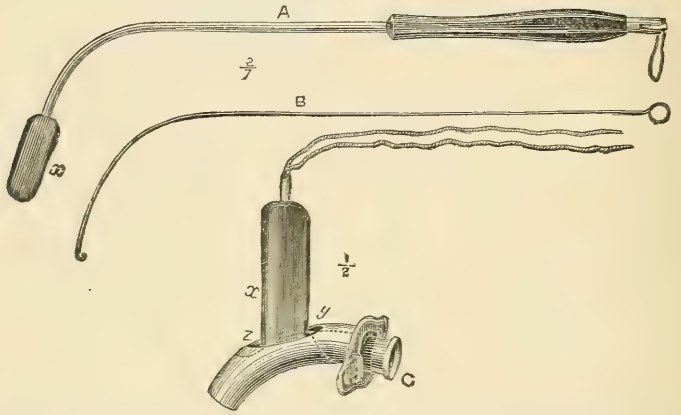


FIG. 62.—Schrötter's Laryngeal Dilator. A, The handle with bougie attached, ready for passing into the larynx; B, a slender rod for drawing the cord through the handle. The bougie *x*, having been passed through the laryngeal stricture, enters the fenestra in the tracheal cannula and is held in place by the inner cannula C, which passes through an opening in the lower end of the bougie.

patient. At the commencement of treatment this will be perhaps not longer than a half-hour, but as the patient becomes tolerant it may be left in place a day or longer, and need only be removed for the purpose of cleansing, or for the insertion of a larger size. After unclasping the spring forceps the bougie can readily be withdrawn by means of the cord.

A modification of the instrument dispenses with the spring forceps, the dilator being held in place by the inner cannula of the tracheotomy tube. This process of dilatation is of necessity a slow one, and many instruments have been invented to forcibly dilate the stricture, the bougie being afterwards inserted to prevent contraction of tissue. Figs. 63 and 64 show Mackenzie's dilator, consisting of three blades bent at a suitable angle, which are introduced closed into the larynx

and opened by turning a screw at the proximal end. An index and dial plate on the handle indicate the amount of distention accomplished.

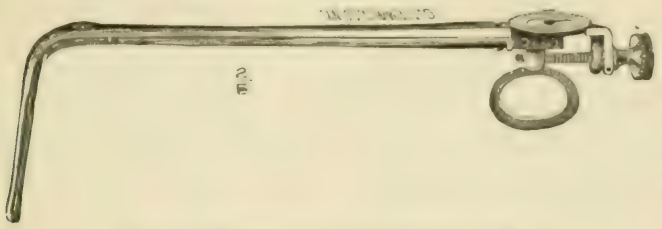


Fig. 63.—Mackenzie's Laryngeal Dilator Closed.

Fig. 65 shows Navratil's dilator, which is formed on a somewhat similar plan. The long tube is bent to the laryngeal curve and con-

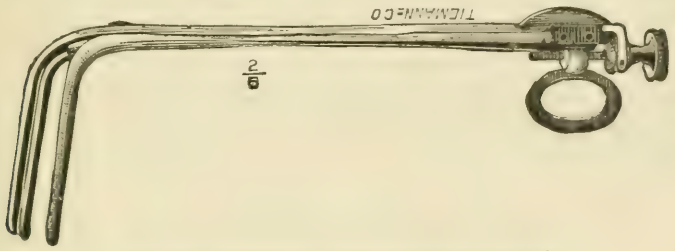


Fig. 64.—Mackenzie's Laryngeal Dilator Opened.

tains a steel rod. On its distal end is an olive-shaped bougie made up of segments which are attached by movable arms partly on the

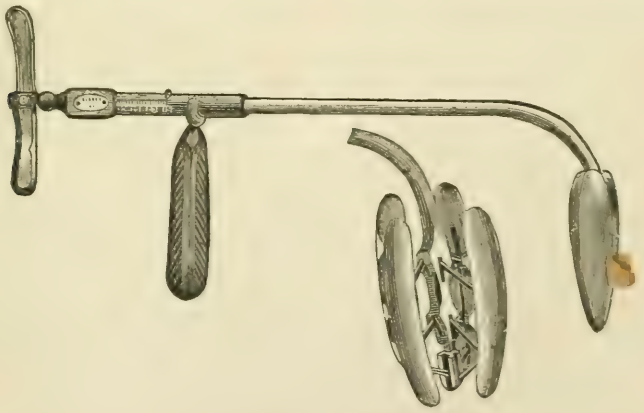


Fig. 65.—Navratil's Laryngeal Dilator.

outer tube and partly to the central steel rod. The patient holds the instrument by a handle which is attached to it; a screw at the proximal end draws the steel rod within the outer tube, and by drawing

forward the movable arms expands the segments. A scale indicates the amount of dilatation.

Störck has devised a three-bladed dilator which is intended for insertion through the tracheal opening, in order to dilate the strictures from below, but I prefer the manipulation through the mouth. Cutting is better than dilatation where there is adhesion between the true cords or the ventricular bands, or where a web has formed across the glottis. A concealed knife, such as Schrötter's or Mackenzie's, in which the blade emerges from a tube after insertion in the laryngeal cavity is safer than a naked knife.

Whistler's cutting dilator puts the tissues on the stretch before they are incised, which is of advantage in rendering the cutting more thorough. An olive-shaped bougie contains a concealed knife, which may be made to protrude anteriorly or posteriorly after the instrument is in place (see Fig. 66). Browne's instrument is smaller and pos-

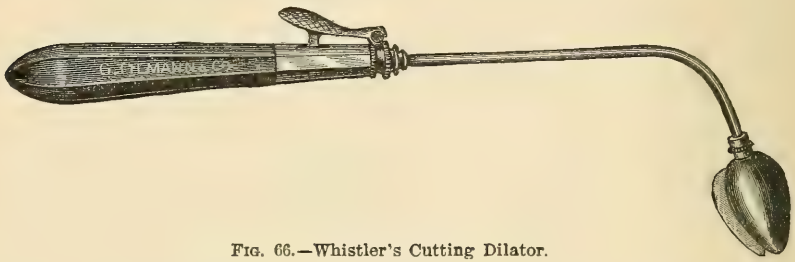


Fig. 66.—Whistler's Cutting Dilator.

sesses a hollow tube through which the patient can breathe while the instrument is in position.

Cutting instruments may be used in all forms of laryngeal stricture as they facilitate dilatation, but the dilators must be employed after incision to prevent adhesion of the cut surfaces. The passage of a catheter through the larynx is easily accomplished, and can be done without previous tracheotomy, therefore it might be of use in syphilitic stenosis in the early stages. Metallic dilators or bougies alone can overcome the resistant character of the cicatricial bands at a later stage. A metallic catheter might serve, but intubation by O'Dwyer's method is certainly far better, it does not require tracheotomy, and a sufficient aperture of the air passages is maintained while the treatment is carried on. Where the stenosis is too great to admit a tube large enough for breathing purposes, O'Dwyer suggests anæsthetization of the patient and forcible dilatation of the larynx until a suitable tube can be inserted. The distortion of the larynx offers the greatest obstacle to the insertion and retention of an ordinary tube. When available, intubation is unquestionably the best method

of treatment of syphilitic laryngeal stenosis, but whatever measure is adopted contraction will undoubtedly recur, and treatment will need to be constantly reapplied.

Tuberculosis of the Larynx.

It is a somewhat curious clinical fact that tuberculous disease manifests an increasing virulence according as the deposit occurs in portions of the mucous membrane nearer to the outer world. Thus pulmonary tuberculosis, with our increased knowledge of its true significance and the improvement in our methods of dealing with it, is at the present day regarded as one somewhat amenable to remedial measures, especially in its earlier stages; when a tuberculous deposit occurs in the larynx, however, we regard this as constituting a more serious affection. If the deposit occurs in the buccal or pharyngeal cavity its import is still more grave; in this latter region, I believe it is to be regarded as a manifestation or forerunner of acute tuberculosis in probably all instances, while when confined either to the larynx or to the pulmonary tissue it usually pursues a chronic course. Moreover, while the natural habitat of tubercle is the pulmonary tissue, the frequency with which deposits occur in the regions above diminishes as we approach the outer world. Thus Williglk in one thousand three hundred and seventeen autopsies of tuberculous cases found the larynx affected in two hundred and thirty-seven instances, while the pharynx was involved in but a single instance. While the older writers undoubtedly observed to some extent the not infrequent occurrence of grave throat complications in connection with pulmonary disease, they seem to have failed entirely to establish any direct connection between the two, and it is not until the seventeenth century that we find any record of an attempt to investigate the true nature of the throat symptoms. Thus in the latter part of the seventeenth century we find Sylvius and Morton recording the existence of nodules in the throat which they seemed to regard as tuberculous, and a hundred years later Stark and Baillie made somewhat similar observations. About this time we find what may be called the first intelligent comprehension of the symptoms in the recognition on the part of Petit, Portal, and Sauvée of the existence of true ulcerative processes in the larynx, and yet they failed to make any distinction between the ulcerations of tuberculosis and those of syphilis. Laennec's famous treatise on auscultation was published in 1819, and it is usually stated that this writer there recorded his observations on the specific character of the laryngeal ulcerations which occurred in connection with pulmonary phthisis, and further stated that the

ulcerative process was due to the breaking down of a deposit of tubercle in the laryngeal mucous membrane. A careful reading of Laennec's work fails to discover any reference whatever to laryngeal tuberculosis, although he records observations on tubercular disease in most of the other organs of the body. In 1824, five years later, we find the first clear statement regarding the laryngeal complications of pulmonary phthisis, when Pravaz made the positive assertion that the ulcerative processes which occur in the larynx in connection with pulmonary disease are the direct result of the softening and breaking down of tubercular nodules. While, therefore, the true nature of laryngeal phthisis was vaguely suggested in 1810 by Baillie, in his researches on pulmonary phthisis, to Pravaz is undoubtedly due the credit of first clearing up this hitherto obscure question. In the year following the appearance of Pravaz's paper, there was published the famous treatise of Louis in which the doctrine was advocated that the ulcerations of the larynx and trachea and parts above which occur in connection with pulmonary disease were due to the eroding influence of the sputum as it passed over the mucous membrane during its expulsion from the diseased portions of the lung. He supported this theory with a most excellent course of reasoning, demonstrating that these ulcerations occurred only in the larger bronchi which emerge from the large tubercular cavities, and, moreover, that the frequency of the ulceration diminished the nearer the oral cavity was approached; this teaching, however, failed of general acceptance. Still later we find Trousseau and Belloc describing a form of tubercular ulceration of the larynx, but still confusing the question in describing also a syphilitic variety of laryngeal phthisis. The subsequent investigations of Andrals, Albers, and others were in the main confirmatory of the teachings of Pravaz, and subsequently we find it to be the generally accepted doctrine among writers that the morbid process which constitutes pulmonary tuberculosis may also locate itself in the laryngeal mucous membrane, and we find Rokitansky and Virchow making the authoritative statement as a fact beyond question that laryngeal phthisis is due to a deposit of tubercle in the laryngeal mucous membrane.

Etiology.

Tuberculous disease develops in the larynx under much the same causative influences as those which govern its location in any other portion of the economy. Of course, the investigations of Koch have shown conclusively that a tuberculous process is due primarily to the lodgment or colonization of the tubercle bacillus. While this is the direct cause from a clinical point of view, it becomes of still more im-

portance to determine why some persons are susceptible to it, while others escape. This can only be explained on what we call, though somewhat obscurely, the tubercular diathesis, and by this we understand that peculiar systemic condition which in the large majority of instances, if not in all, is the result of heredity. Perhaps no fact in clinical medicine is more thoroughly established than that the descendants of tuberculous parents are peculiarly susceptible to the disease. What physical condition constitutes this susceptibility can only be a matter of speculation. Shakespeare has suggested that it may be due to abnormally wide lymph channels which admit of the entrance of the bacillus. Whether this or some other systemic peculiarity is the source of this susceptibility, pathological investigation heretofore has failed to demonstrate, and we merely accept the existence of this special diathesis as a clinical fact. As a matter of observation, a tubercular deposit in the larynx in the very large majority of instances succeeds or accompanies a similar process in the lungs. It has long been an interesting subject of discussion as to whether the larynx may be affected primarily before the lungs or any of the other tissues of the body. I find in recorded literature many instances in which this is said to have occurred. On the other hand, it is asserted that the laryngeal affection is always secondary to a previously existing morbid condition of the lungs, and that where the disease is said to have manifested itself primarily in the larynx the pulmonary process was latent or still existent and escaped the detection by ordinary physical signs. From a practical point of view, it is difficult to understand the importance of this question. Tubercular deposit may occur in any region of the body primarily; why, therefore, the larynx should be exempt from this primary invasion it is not easy to say. That the larynx may be the seat of tubercular deposit before any other portion of the air tract has been clearly shown by Demme, who reports the case of a child four and a half years of age dying of tubercular meningitis in which the autopsy revealed tubercular deposits in the larynx while the pulmonary tissue was normal; laryngeal ulceration had been observed before death by laryngoscopic examination. While the immediate cause of the disease, therefore, is the lodgment of the tubercle bacillus, the predisposing cause is to be found either in the tubercular diathesis, as it occurs in the large majority of instances, or in previously existing pulmonary tuberculosis. While the theory of Louis that the ulcerative process may be excited by the eroding action of the sputum passing over the laryngeal mucous membrane from the diseased parts below is endorsed by but few observers at the present day, yet it hardly seems wise absolutely to reject the view in endeavoring to explain why laryngeal inflammation occurs in

many cases of pulmonary phthisis. In order that this auto-inoculation should occur it is necessary that this bacillus-laden sputum should pass over an eroded mucous membrane. Now, the most frequent site of an erosion of the larynx is at the posterior insertion of the cords and on the anterior face of the arytenoid commissure. Clinical observation teaches us that in by far the largest proportion of cases this is the point where laryngeal phthisis commences, hence it would seem that a certain amount of importance must be attached to this auto-inoculation theory. In most instances, however, it is probable that there is a common cause for both the laryngeal and pulmonary affections rather than that they stand in any direct causal relation one to the other. The question arises, Does the existence of a tubercular process in the larynx, as recognized by a laryngoscopic examination, necessarily involve the certainty of a coexistent process in the lungs? I think not. As a matter of clinical observation, in the large majority of cases where the laryngeal disease is met with, it occurs in connection with a pre-existing pulmonary affection. Moreover, then, if at the time that the laryngeal affection is discovered no evidences of pulmonary involvement can be detected by ordinary physical signs, we must accept the laryngeal involvement as evidence that the pulmonary tissues are seriously threatened, and also that the development of the diseased action there will very soon manifest itself. In the minds of the laity there is perhaps a somewhat undue gravity attached to laryngeal affections; especially is this true where the voice is impaired in any way. If now such an attack occurs in an individual belonging to a consumptive family, the question arises as to how far the gravity of such a simple catarrhal condition may be increased by this hereditary tendency. Both from a pathological and clinical point of view, I doubt if there is any relation whatever between a catarrhal and a tuberculous process, and I am disposed to regard the influence of a simple catarrhal laryngitis as a predisposing cause of tuberculous disease as greatly over-estimated. The only way, I think, in which a catarrhal process can lead to a tubercular deposit is by the occurrence of erosions in the larynx; that these may occur in chronic laryngitis is possible, but they are certainly exceedingly rare. The increasing disposition on the part of the laity and of the profession to regard tuberculous disease as contagious lends additional weight to this consideration, since the larynx, of course, is more exposed to the lodgment of floating disease germs than the pulmonary structures; and, moreover, I think we must regard the mucous membrane in a state of inflammation as supplying a more favorable nidus for the development of a disease germ than one in perfect health.

Laryngeal phthisis occurs more frequently in males than in

females. It is essentially a disease of adult life, in most instances being met with between the ages of twenty and forty. In 500 cases observed by Mackenzie 35 occurred before twenty; 194 between twenty and thirty, or in the third decade of life; 162 in the fourth decade; and 109 between forty and sixty. Of these, 365 were males and 135 females. Heinze met with 5 cases in the first decade of life, 23 in the second, 130 in the third, 112 in the fourth, 67 in the fifth, 37 in the sixth, 9 in the seventh, and 3 in the eighth, the proportions of males to females being as 14 : 9.

As regards the frequency of the development of laryngeal disease in connection with pulmonary affection, Mackenzie found in 100 cases of phthisis pulmonalis 33 in which the larynx was involved, while Louis found 65 cases of laryngeal involvement in 193 subjects. In Heinze's 1,226 cases of phthisis observed at the Pathological Institute in Leipsic, laryngeal ulcerations were found in 376 cases. We thus find the percentage holding pretty steadily from 30.6 per cent. to 33 per cent. On the other hand we find that Williglk in 1,317 autopsies observed but 237 instances of laryngeal involvement, or 13.8 per cent., while Schrötter found but 42 cases in 723 autopsies, or a trifle over 6 per cent. It is not easy at first sight to reconcile these statistics. In Mackenzie's 100 cases he reports that the larynx was absolutely healthy in but 29 instances, while in 20 there was infiltration, and true ulceration in but 13—in other words, there was tuberculosis in but 13 per cent. While, of course, we cannot call in question the accuracy of such careful observers as Louis and Heinze who have shown over 30 per cent. of cases with laryngeal disease occurring in phthisis pulmonalis, we draw the conclusion that if all cases which showed any form of tubercular deposit in the larynx were incorporated in these statistics, careful pathological investigation by autopsies would show laryngeal involvement in from 30 to 33 per cent. From a clinical point of view I am disposed to regard Williglk's statistics as according more closely with what we may expect to meet with in ordinary practice, and that this percentage of cases of pulmonary tuberculosis may be expected to develop active disease of the larynx, viz., that form of laryngeal phthisis which manifests itself by well-marked subjective symptoms and in which the disease process in the larynx is characterized by a progressive waste of the tissues.

Pathology.

From a practical point of view, I think we must regard a tuberculous process in this region as an inflammatory process possessing certain peculiarities, being primarily dependent upon and probably modified by the presence of the specific bacillus of tuberculosis. In

the early stages of the process we find the membrane infiltrated with a large number of small, rounded embryonic cells, which, while they diffuse themselves somewhat actively through the mucous membrane proper, show a tendency to gather in small and somewhat circumscribed masses. These circumscribed masses are supported by a delicate reticulum; they are more densely crowded toward the periphery, while the central portion of each collection shows a tendency to necrosis, as is evidenced by the presence of granular matter. In many instances the centres of these collections are found to be occupied by rounded protoplasmic masses containing large numbers of nuclei, and which show on their periphery slender, thread-like processes, these constituting the giant cells.

The primary deposit occurs in the mucosa proper, the epithelial layer being in no way involved. The greatest activity of the process occurs about the walls of the blood-vessels, and to a somewhat less extent in the neighborhood of the glandular structures. Where they invade the blood-vessels the walls also are attacked and are seriously encroached upon, or even completely obliterated. The tuberculous process in the larynx, probably owing to its constant activity, is a progressive one and the tendency is to develop into an ulcerative process. As we have seen, post-mortem examinations show a much larger number of instances where the larynx is infiltrated than where ulceration has resulted. We must explain these cases on the ground that death has supervened on account of the advanced stage of the pulmonary disease before the laryngeal disease had time to develop into an ulcerative process, although that a tuberculous deposit may remain quiescent for a considerable period of time cannot be questioned.

The immediate cause of the ulceration in this process is undoubtedly the excessive crowding together of the rapidly proliferating cells, thus giving rise to localized necrosis by the breaking down of the tissue in the centre of the circumscribed masses or nodules. This breaking down and discharge of the nodule establishes a small point of ulceration on the surface of the mucous membrane, which by a slow process of extension widens its borders. In this way a number of points of ulceration coalesce and form a broad and more or less extensive ulcerated surface. The progressive character of the process is still further shown by the invasion of the parts beneath the mucosa proper. In this way the perichondrium may be involved and the affection thus become complicated by an attack of perichondritis, leading ultimately to a necrosis of the cartilage. In the same way the muscular structures and fibrous tissues of the organs may be successively attacked.

In the majority of instances the first manifestation of the affection consists in a circumscribed deposit usually on one side of the organ; the most frequent location of this is in the mucous membrane covering the arytenoid cartilage or the commissure. Next in frequency we have involved the mucous membrane covering the aryepiglottic folds, the ventricular bands, the true cords, and lastly the epiglottis. Following the primary invasion, the other portions of the larynx may become involved by lateral extension, or by the development of new centres of infiltration. After the process has commenced on one side of the larynx, the other becomes involved, either by lateral extension or by auto-inoculation—a method which seems to have been observed in a number of instances reported in literature. In many instances we observe what appears to be an œdematous condition of the mucous membrane of the larynx as the result of a tubercular infiltration, but this may be due to invasion of the perichondrium, although oftener it is probably the result of the walls of the veins becoming involved whereby their lumen is encroached upon and the return circulation of the blood is seriously hampered or arrested. It is to be borne in mind, however, that the tubercular infiltration of the mucous membrane itself leads to appearances which to no small degree resemble, without constituting, an œdematous condition.

Symptomatology.

As we have seen, the primary invasion of tubercle occurs in the arytenoid commissure, thus preventing approximation of the cords, hence impairment or loss of voice is liable to be one of the earliest symptoms calling attention to the laryngeal involvement. A chronic laryngitis of simple catarrhal character not infrequently occurs in connection with pulmonary phthisis; this, however, gives rise merely to a hoarseness, the voice being harsh and of course with lowered pitch. In tuberculous disease the vocal impairment shows itself in its soft, weak, and somewhat aphonic character; at the same time there is liable to be a certain amount of what we call phonatory waste, approximation of the cords being prevented, the glottis is abnormally wide in the effort at phonation and an undue amount of air escapes in the effort of talking, which thus becomes labored and wearisome. Moure calls attention to a symptom in this affection which is peculiar, in that the voice becomes what he calls "bitonal": while the patient talks in a low tone, the voice suddenly breaks into a falsetto note which is maintained for a short time and then the lowered pitch is resumed.

A moderate amount of extension of the infiltration to the posterior insertion of the cords is not infrequent, giving rise to a hoarseness

which is due to interference with the vibration of the cords; but the prominent characteristic of the voice is still that of weakness, though it is somewhat harsher in character than when approximation is merely interfered with. As the deeper portions of the larynx are invaded the muscular structures become weakened; thus the articulations are involved in such a way as to render the voice completely aphonic, and the patient converses in a soft, weak whisper. In rare instances the voice may remain unimpaired up to a late stage of the disease. In such a case, of course, the tubercular infiltration confines itself to the upper portion of the larynx. Mackenzie reports the voice as having been unaffected in forty out of five hundred cases, which would seem a somewhat large proportion.

Painful deglutition sets in very early in the history of the disease and is liable to become a very serious feature and at times it is of all the most distressing one. Where the arytenoids alone are involved this is due to pressure on the epiglottis in the act of deglutition or possibly to the crowding of the bolus of food, and it is not of a very distressing character. It may occur even in the primary stage of infiltration, but when ulceration occurs the pain becomes more severe in character, increasing with the extent of the swelling and ulceration.

With the involvement of the epiglottis there sets in a train of symptoms which render the condition of the patient pitiful beyond description, especially after ulceration has commenced; the parts become so exquisitely sensitive that the act of deglutition becomes not only difficult but exceedingly painful; so great is the suffering which the taking of food involves that the swallowing of solids becomes impossible, and even the attempt at swallowing fluids gives rise to such acute distress that the patient becomes averse to making the effort. With the loss of tissue which the ulcerative process involves the epiglottis fails of its duty, and portions of food may make their way into the larynx, giving rise to acute attacks of coughing, or, what is still more distressing, to attacks of choking, or spasm of the glottis. Furthermore, as the result of the immobility of the parts which the infiltration causes, particles of food, and especially fluids, make their way into the nose during the act of deglutition.

Cough is invariably present, and while in large part it may be due to pulmonary disease, its severity and persistency are greatly aggravated by the laryngeal complication; this is especially true during the ulcerative stage of the affection when the secretions from the ulcerating surface, which are thick, ropy, and tenacious, accumulate in the larynx and prove an additional source of irritation, in that the patient finds it exceedingly difficult to dislodge them.

Tenderness and pain on pressure are always present, but subjec-

tive pain is not usually characteristic of the first stage. The secretion from a tubercular ulcer is not large in amount but is of a thick, inspissated, semi-transparent, ropy mucus, which clings closely to the parts and is dislodged with great difficulty. If the secretion is profuse and purulent or muco-purulent in character this should be recognized as having its source in the pulmonary disease. Hæmoptysis is in no way to be regarded as a symptom of laryngeal phthisis except as far as minute amounts of blood are lost as the result of injury from the passage of irritating masses of food; free hemorrhage from the larynx I have never met with. If the perichondrium is involved and perichondritis results, this does not prominently complicate the disease, in that the tuberculous form of perichondritis presents none of the markedly painful symptoms which usually characterize this affection. Necrosis of the cartilage is the usual result. The sequestrum may remain embedded in the tissues or may be expelled. While perichondritis does not add any very special painful symptoms to the disease, it may cause such an amount of swelling as notably to encroach upon the lumen of the larynx, giving rise to dyspnœa. In several such instances tracheotomy has become necessary. A moderate amount of œdema is occasionally present, but rarely to such an extent as to cause dyspnœa. That death may result from this cause is attested, as far as I know, by a single case; while, therefore, œdema and perichondritis may give rise to dyspnœa in laryngeal phthisis, this is exceedingly rare. I think that a more common cause of dyspnœa is the narrowing of the glottis from ankylosis of the crico-arytenoid articulation. Curiously enough when this happens the fixation usually occurs with the cords in or near the median line, giving rise to a condition not unlike that of bilateral paralysis of the abductor muscles. Tubercular infiltration of the epiglottis results in ulceration and destruction of tissue rather than in perichondritis, this cartilage being a fibro-cartilage.

The cachexia which manifests itself in these cases we recognize as characteristic of the pulmonary disease upon which the laryngeal affection is superimposed, and yet the invasion of the larynx has seemed to add an additional element to the general systemic impression—more, proportionately, than is to be explained by the area of tissue involved. This is due in part, perhaps, to the greater physical pain and suffering which tubercular laryngitis involves, together with the interference with proper nutrition on account of the pain which accompanies the act of deglutition. There is a still further element of depression in the dread and apprehension which exists in the minds of most people suffering from throat consumption, for while not admitting it, I think these patients are fully conscious of the fact

that, the disease having attacked the larynx, the prognosis is rendered exceedingly grave thereby. While the emaciation and loss of flesh occurring in laryngeal phthisis can be explained by the existence of pulmonary disease, I think those of us who see many cases of this disease learn to recognize a certain pain-drawn, anxious facial expression as almost characteristic of throat consumption.

Diagnosis.

The development of mere hoarseness in the course of a pulmonary tuberculosis does not necessarily imply a tubercular invasion of the larynx, but if this vocal impairment is attended with subjective symptoms of pain in deglutition and tenderness on pressure, the symptoms point pretty clearly to the grave character of the complication. The mere existence of Koch's bacillus in the sputa, upon which we depend so much nowadays for our diagnosis of pulmonary phthisis, gives us no information as to the special site in the air tract which has been invaded. An absolute diagnosis, then, of laryngeal phthisis will depend necessarily upon the laryngoscopic examination. We have the authority of von Ziemssen for the statement that a tuberculous process in the larynx cannot be definitely recognized as such by ocular inspection in those cases in which the lungs are also invaded. On the contrary, I am of the opinion that a tuberculous process presents appearances so characteristic and typical that a failure to recognize it by one having any experience in laryngoscopic work would be scarcely excusable. While, perhaps, I will not go so far as Browne, who states that "we know no disease in which, with the laryngoscope, we can be so sure of our diagnosis," I think the cases are very rare in which an error of diagnosis need be made. As we have already seen, a tubercular infiltration invades the mucosa proper and also shows special affinity for the muciparous glands and the blood-vessels, the calibre of the latter being diminished, hence we have as a result three phenomena: first, tumefaction; second, exsanguination; and, third, diminution of secretion. A laryngoscopic examination, therefore, shows us in some portion of the larynx, and in the large majority of cases this is in the arytenoid commissure, a peculiar, pale-colored tumefaction, which in appearance and color is almost pathognomonic of tuberculous disease. It is of a pale pinkish color, with a grayish tinge, rounded in outline and presenting a dry, glassy surface. When this invades the region of the arytenoid it gives rise to what we generally term club-shaped arytenoids. This usually constitutes the first stage of the disease, viz., that of infiltration. Occasionally this tumefaction is more marked on one side than on the other, but in the large proportion of cases it is uniform, when

involving the arytenoid region. If it invades the ventricular bands or the aryepiglottic folds it is more liable to be unilateral. If the epiglottis is invaded the disease rapidly extends to its crescentic edge, giving rise to what is usually termed the turban-shaped epiglottis (see Fig. 67). In this region, while at its onset the tumefaction may be unilateral, it very rapidly assumes a symmetrical shape, invading both sides. While at the very onset of the attack the appearances are those of a mild inflammation with the element of hyperæmia to a certain extent absent, we very soon see a new appearance, in that the mucous membrane becomes dotted over or studded with minute yellowish points, which mark the development of the tubercular nodules nearer to the surface of the membrane. They give rise to no

FIG. 67.—Tuberculosis of the Larynx, with Infiltration of the Epiglottis, producing the so-called "Turban-Shaped" Epiglottis.

FIG. 68.—Tuberculosis of the Larynx. Numerous miliary tubercles are seen lying just beneath the epithelial layer of the mucous membrane.

observable elevation and yet are clearly seen through the transparent epithelium of the surface (see Fig. 68). These spots are of a grayish-yellow tinge and of lighter color than the mucous membrane surrounding them.

The further development of the disease consists in the breaking down of these minute points or nodules, and the establishment of small ulcers which now increase by the extension of their borders, and finally coalesce with their neighbors, forming a broad area of ulcerative action. With the establishment of this third, or ulcerative, stage of the disease the whole aspect of the tissues changes, but the affection still maintains a characteristic appearance which is scarcely to be mistaken for that of any other morbid process with which I am familiar in the larynx. What now occurs is a destructive process: the epithelial layer is exfoliated and there is revealed the tuberculous pro-

cess in the mucosa proper. The color of the tubercular ulcer is not markedly different from that of the mucous membrane of the stage of infiltration, it still preserves its grayish-yellow appearance, although we may observe it occasionally dotted over with minute reddish points, small blood-vessels eroded or ruptured by the morbid process. The surface is ragged and worm-eaten, as it were (see Fig. 69). Fraenkel likens the surface of the tubercular ulcer to cut bacon; La Boulbène to the track of earthworms in the wet sand. It has always seemed to me not unlike a dish of wet meal which birds have pecked at. The ulcerated surface as well as the mucous membrane surrounding it are of a grayish, pasty color and notably anæmic, the two resembling each other so much that at first sight it is not always easy to determine just where the ulcerated surface terminates and the unbroken mucous membrane begins. The edges, moreover, are irregular in outline, and flush with the surrounding tissues. We thus have the curious phenomenon of a superficial waste of tissue without any apparent loss of contour; this is due to the fact that the tubercular infiltration in the tissues beneath goes on so rapidly as fully to compensate for the superficial waste. The loss of tissue, therefore, is not apparent in that the general contour is to a certain extent maintained as regards bulk, although the normal outlines of the larynx are seriously disorganized.

FIG. 69.—Extensive Tubercular Ulceration of the Larynx.

The secretion from the tubercular ulcer is limited in extent and consists of a thick, ropy, tenacious mucus rather than pus. It is grayish in color and presents practically no evidence of the presence of the pus corpuscle, or necrotic tissue. While we make the statement that the result of ulcerative action is no apparent loss of tissue, we might perhaps make an exception in the case of the epiglottis, which being composed of fibro-cartilage does in certain cases become the seat of destructive action and we may have the whole of the upper portion of it destroyed; but still while its contour is practically destroyed, the mass remains, as the result of infiltration, much larger than the original cartilage. The same is true, although to a less extent, of the vocal cords. The arytenoid cartilages occasionally become necrosed and exfoliated as the result of the tubercular invasion of the perichondrium; even this, however, does not result in any

apparent loss of tissue. The point which I desire to emphasize in this connection is that, however extensive the destruction of tissue may be superficially, and however completely the original contour of the larynx may be destroyed, there is still the extensive infiltration of the parts beneath which results in a thickening, more than compensating for the loss of tissue which accompanies the ulcerative process.

As I have already intimated, I regard tubercular disease as presenting for ocular inspection appearances so characteristic and typical that a mistake in diagnosis should not be made as a rule, and yet, perhaps, the discussion of the question would be incomplete were we not to emphasize certain points which may aid us in the differential diagnosis between it and syphilis, lupus, perichondritis, and malignant diseases.

In the superficial ulcer of syphilis we have a discharge which is distinctly yellow and more or less profuse in character; a rounded outline, a slightly excavated surface, and an areola limited in extent, although notably reddened but not swollen. In tuberculosis, on the other hand, we have a limited amount of secretion of gray, ropy mucus; there is no excavation, and there is no areola, the mucous membrane surrounding it being swollen, infiltrated, and notably exsanguinated.

In the deep ulcer of syphilis we have all the characteristics of the superficial ulcer exaggerated; there is deep excavation and more or less profuse discharge mingled with necrotic tissue; there is a markedly injected area, differing again, as may be seen, in every way from the tuberculous process, although, of course, it is to be borne in mind that the two processes may occur simultaneously in the same larynx, numbers of instances of which have been recorded in literature. When this occurs, however, each disease seems to maintain its characteristic features.

In lupus we have the nodular swelling, very limited amount of hyperæmia, and the almost total absence of secretion of any kind. We call it ulcerative action, but it is by no means easy to recognize it as such at first sight by laryngoscopic examination.

Perichondritis may possibly be mistaken for the infiltration of the tuberculous process, but not for the ulcerative stage. The notably exsanguinated appearance of the tuberculous lesion presents a totally different aspect, however, from the highly injected and markedly irregular swelling which characterizes the inflammatory disease.

In malignant disease of the larynx we have unilateral tumefaction, irregular nodular outline, and a highly injected mucous membrane, and where ulceration occurs there is more or less profuse secretion

of ill-smelling muco-pus, often charged with blood and necrotic tissue. Laryngeal stenosis, moreover, is the rule in malignant disease, and an exceedingly rare occurrence in tuberculosis.

As already stated, one of the early symptoms of the disease is vocal impairment, the voice assuming a soft, weak character, differing essentially from the voice of catarrhal laryngitis or of specific disease. This vocal impairment in laryngeal phthisis may be due to infiltration or ulcerative action involving the vocal cords, but in the majority of cases it is due to infiltration of the arytenoid commissure, by which the approximation of the cords for phonative purposes is interfered with. In rare instances the impairment of mobility of the cords may be due to an invasion of the crico-arytenoid articulation, giving rise either to impaired mobility or to complete ankylosis. This may be unilateral or bilateral. It is a somewhat curious fact that when ankylosis of this articulation occurs, it results in a fixation with the cords in adduction. A laryngoscopic examination when this condition occurs will reveal the cords apparently motionless either in or near the median line, or in the cadaveric position. A number of cases have been reported in literature of bilateral paralysis of the abductors with recurrent attacks of dyspnœa in connection with laryngeal tuberculosis, in which it seems to me probable that the apparent paralysis may have been due really to ankylosis of this joint as a result of tuberculous invasion. Cartaz has reported two cases in which this form of pseudo-paralysis was the first manifestation of laryngeal disease.

Prognosis.

When in the course of a pulmonary phthisis a laryngeal invasion supervenes, it is to be regarded as an exceedingly serious complication, and one which adds an additional gravity to an already unfavorable prognosis. When the disease develops primarily in the larynx, I think we are justified in regarding it, on the lines already laid down, as evidence that the tuberculous infection is characterized by an unusual virulence and activity.

The average duration of life in pulmonary consumption is generally stated as about three years. In Mackenzie's one hundred cases of laryngeal phthisis he deduced the fact that in the greatest number of instances death resulted in from twelve to eighteen months after the occurrence of the throat symptoms, and that in sixty-six per cent. a fatal issue occurred between six months and two years. These statistics fail to show us the average duration of life in cases of pulmonary tuberculosis which become complicated with laryngeal disease. In my own recorded cases of laryngeal phthisis I find that the longest

period which any patient survived after the commencement of pulmonary trouble which was subsequently complicated by laryngeal invasion was forty-six months, while the shortest time was three months, the average duration of life being twenty-two and three-quarter months. This, I think, indicates with a fair degree of correctness the average duration of life in laryngeal phthisis, estimating from the time of the first manifestation of tuberculous disease, whether in the lungs or in the larynx. In other words, if the average duration of life in pulmonary phthisis is three years, the occurrence of laryngeal complications so far indicates an additional virulence of original infection, and adds so far to the gravity of the attack, that the probable duration of life is reduced thirty-three per cent. To repeat briefly—the average duration of life in an ordinary attack of pulmonary consumption is three years; the average duration of life in pulmonary consumption complicated by a laryngeal invasion is two years; the average duration of life after the supervention of laryngeal invasion is eighteen months.

In individual cases the prognosis must necessarily be influenced by special circumstances. The most important of these is the progress of the pulmonary disorder. If the laryngeal invasion occurs in the later stages of a pulmonary disorder it will necessarily run a rapid course. On the other hand, if it occurs during the first stage of the pulmonary phthisis and does not advance rapidly, it may run an exceedingly chronic course. This is especially true, I think, with reference to those cases of laryngeal invasion which occur in connection with fibrosis of the lungs. The seat of primary deposit in the larynx, I think, has a marked influence upon the progress and development of the disease. If the epiglottis is attacked the subjective symptoms, as already indicated, become of an exceedingly painful character, while at the same time the laryngeal process seems to be markedly accelerated and ulceration sets in early owing to the fact that the epiglottis is constantly impinged upon and irritated by the bolus of food in the act of deglutition. In a less degree the same holds true when the mucous membrane covering the arytenoid commissure and cartilages is involved. Clinical observation shows us that when the primary invasion occurs in one of the ventricular bands or vocal cords—a portion of the larynx not subjected to much pressure or functional activity—we may find the lesion develop rather slowly. From a practical point of view our most important consideration in this connection has to do with the prognosis as regards the success of remedial measures. Unquestionably the very large majority of cases which come under our observation fail to respond in any notable degree to any of our remedial efforts. I think we are

justified, in the discussion of this question of prognosis with reference to treatment, in confining ourselves entirely to the laryngeal manifestations, and may fairly claim a cure in any given case if the tubercular process in the larynx is arrested, even though the pulmonary disease persists. In this view I think we may go still further and say that patients have rarely died of laryngeal phthisis, as the laryngeal disease is a complicating lesion and the direct cause of death is the pulmonary disease. If our remedial efforts are successful in arresting the local lesion in the larynx, we not only prolong life but relieve our patient from the grave complication of the pulmonary disorder, which is the source of an amount of suffering and distress that is equalled by very few diseases which we are called upon to treat. If this view is tenable I am disposed to think that the prognosis of laryngeal phthisis is not so unfavorable as is generally claimed; for while undoubtedly, as already stated, a number of cases resist all treatment, in a certain proportion of instances we may entertain a reasonable hope of arresting the morbid process by a carefully carried out and judicious course of general and local treatment, and even if we fail to arrest the disease, we certainly can control, to a very large extent, the more distressing symptoms, thus relieving suffering and adding to the comfort of the patient. This is especially true of the early stage of the disease before ulcerative action has set in. By far the gravest development that occurs in laryngeal disease is the invasion of the epiglottis. When this occurs it is questionable if we can accomplish more than to relieve the more distressing symptoms, although once certainly, in my own experience, I am confident that I have seen a case in which the epiglottis was involved, in which prompt measures, taken immediately upon its invasion, and before ulceration occurred, served to arrest the disease.

I find reported in literature a number of instances of spontaneous cicatrization of tuberculosis, and yet in no form of ulcerative action in the air tract are we able to detect in a less degree, any reparatory effort on the part of Nature than in tuberculosis.

Treatment.

Up to comparatively recent years the routine treatment of all forms of ulcerative action, including tubercular, syphilitic, and others, confined itself largely to the use of nitrate of silver, either in the solid stick or one of the stronger solutions. Not only were these applications exceedingly painful in the tubercular ulcerations which are exquisitely sensitive, but it is doubtful if their action was anything more than ephemeral at best, and in many cases undoubtedly more harm than good was accomplished. The natural reaction which fol-

lowed the abandonment of these severe measures led to an entirely opposite procedure, which confined itself mainly to the use of mild palliative and soothing remedies. The importance of this mild plan of treatment was first emphasized by the writer some fifteen years ago and a definite plan laid down, the success of which was shown by a series of reported cases.

This plan consisted of four steps as follows: First, the thorough cleansing of the ulcerated surfaces; second, the application of a mild astringent; third, the application of an anodyne; and fourth, the application of iodoform. The cleansing is accomplished by the use of a carbolized alkaline solution, of which perhaps Dobell's answers the best purpose. This is to be applied by means of an atomizer and preferably with the compressed-air apparatus, with sufficient force to dislodge the tenacious mucus, thereby effecting a thorough cleansing of the parts. The second step consists in the application of an astringent; in the order of preference these are: Sulphate of zinc gr. v. to the ounce; nitrate of silver, gr. iij. to the ounce; chloride of zinc, gr. iij. to the ounce. The third step consists of the application of morphine by means of the spray in the form of Magendie's solution, bearing in mind always that the absorption in this region is quite as active as in the stomach, and hence no more than the physiological dose should be applied. Immediately following the morphine application a ten-per-cent. solution of cocaine should be applied. After this a powder of iodoform may be thrown in, or, better still, europhen in solution as in the following:

℞ Creosote, ... gr. i.
Europhen, ... gr. x.
Albolene, ... ʒ i.

Europhen has much the same action as iodoform and has none of its disagreeable odor.

This plan of treatment should be repeated every second or third day, or, if the case is an aggravated one, daily applications will be necessary. In addition to this I think it is well to put in the hands of the patient for use during the intervals of treatment, especially in those cases where ulceration has occurred and deglutition is painful, a hand-bulb atomizer, by means of which the following inhalations can be given three or four times daily, and especially immediately before eating:

℞ Morphine sulphate, ... gr. iv.
Cocaine muriate, ... gr. xx.
Aqua, ... q. s.
M. ft. sol. et adde
Albolene, ... ʒ i.

In the stage of infiltration I have rarely seen a case in which this plan of treatment failed to give notable relief to the subjective symptoms, and in very many instances an observable improvement was remarked upon a laryngoscopic examination.

After ulceration has developed we have to do, of course, with a much graver lesion, and yet even while we cannot feel so hopeful as regards arresting the disease we may always have entire confidence in our ability to relieve very largely its distressing features, and in a certain proportion of the cases retardation if not an improvement in the local process may be anticipated. Of course, it is scarcely necessary to say that the cases which yield most promptly are those in which the local process is confined to the ventricular bands and the vocal cords, or to the commissure.

The value of europhen or iodoform, of course, is largely in the ulcerative stage, and its importance here deserves emphasis; thus Lublinski reports seventy-five cases treated by iodoform, of which seventy-three were improved and two cured; while Simon reports all his cases improved by this preparation.

The inhalation of medicated vapors by means of the steam atomizer or of the volatile oil of tar, iodine, creosote, benzoin, eucalyptus, and drugs of that sort, have been largely recommended in the treatment of this affection, as in other throat disorders, but I have never seen any benefit result from their use. As regards the use of hot steam applications or inhalations, I am disposed to think they are decidedly objectionable. Some fifteen years ago a most original and somewhat daring plan of treatment was suggested by Schmidt, who advises that wherever the parts were swollen and œdematous, free incision should be made directly through the tissues in such a way as to allow the free escape of blood and serum. I have never felt justified in resorting to this scarification in tubercular disease and to me it seems a somewhat hazardous method, although Schmidt reports excellent results from his operation, as regards relief to the local symptoms. A cure by this plan has not been claimed.

Krause some ten years ago introduced the lactic-acid treatment in tubercular ulcerations of the larynx. The use of this remedy was suggested by the excellent results which Mosetig-Moorhof had obtained in the treatment of lupus vulgaris, caries fungosa, and superficial epithelioma. Krause's method consists of rubbing the lactic acid thoroughly into the diseased surface by means of a cotton pledget or by injecting it by means of the hypodermic syringe. He commences by the use of a ten-per-cent. solution and gradually increases the strength up to an eighty-per-cent. solution, making daily applications until local necrosis occurs. Krause reports a series of cases of

laryngeal phthisis in which this plan of treatment was followed either by a cicatrization of the ulcer or by a marked improvement in the local condition. Heryng advises a thorough curetting of the ulcerated surface by means of a sharp spoon, thus removing as much of the diseased tissue as possible, and after this he rubs in his solution of lactic acid. He reports having succeeded by this plan in securing complete cicatrization in eight out of twelve cases. These patients subsequently died of their pulmonary disease, yet the laryngeal complication remained cured for periods varying from two to seventeen months. Heryng records another series of fifteen cases of laryngeal phthisis in which Krause's treatment of lactic acid alone was pursued, in which he reports eleven cures. I have seen more or less of this surgical treatment of laryngeal phthisis and have had an opportunity of observing some of the cases which were under this method, and while my own personal experience is not sufficient for me to speak authoritatively on the subject, yet it has always seemed to me a somewhat harsh course to pursue; but still in view of the notable success which Heryng, Krause, Gleitsmann, and others have reported to have resulted, I do not think one is justified in condemning it. Rosenberg reports fifty-seven cases of laryngeal phthisis in which nine were cured by the daily application of a twenty-per-cent. solution of menthol in olive oil, the solution being dropped on the diseased surface by means of a small syringe or applied by an atomizer. He directs that the patient also use inhalations of the same, a few drops being volatilized by boiling water. A number of writers advise the use of the galvano-cautery. I quite agree with Isambert, who regarded the use of caustics as highly objectionable, and of these I know of none more so than the electric cautery in laryngeal phthisis.

Occasionally œdematous swelling of the parts, or extensive infiltrations with a complicating œdema, may so far encroach upon the breathing space as to render tracheotomy necessary. If this measure is demanded for the relief of dyspnœa, of course, its propriety cannot be questioned. Usually a very marked relief to local symptoms is immediately observed to follow. This result has suggested the question whether the operation might not be performed as a direct remedial measure. Indeed, among its advocates we find such names as Dupuytren, Krishaber, Fleury, Schmidt, and others. The ground on which it is recommended is, of course, the complete functional rest which the larynx thereby acquires. Schmidt claims that in addition a more complete oxygenation of the blood is secured by the free entrance of air through the tracheal tube.

Mackenzie, Moure, and Cohen speak with absolute disapproval of

this measure, the former stating that its effect is only to prolong a miserable existence, while the latter regards it as in no way, neither directly nor indirectly, curable. In two cases in which tracheotomy was done under my own observation there seemed to be a notable relief from both pain and dysphagia, but further than this the operation was useless, in that life did not seem to be in any way prolonged, although in fifteen operations done by Schmidt he claims that there was notable prolongation of life in five instances. Ripley also reports a case in which not only was suffering relieved, but life was prolonged by opening the trachea. While, therefore, we are scarcely justified, as the result of clinical observation, in recommending this measure as one to be adopted in the ordinary class of cases, yet certainly its promise of relief to local symptoms is to be borne in mind where we meet with instances in which the local symptoms assume a particularly distressing character. The introduction of Koch's lymph in 1890, as a remedy which possessed the property of controlling a tuberculous process, was hailed with no small degree of enthusiasm; the results of observation, however, soon demonstrated that the claims made for it were greatly over-estimated, and an extensive use failed to show that it possessed any of the curative properties which were claimed for it. Its introduction remains now simply as an interesting episode in the industrious search for some remedial measure which shall control the ravages of this hitherto fatal affection.

As to the results of the introduction of the cantharidate of potassium, advocated by Liebreich, of the chloride of gold and sodium and of the iodide of manganese, advocated by J. Blake White, and the rectal injections by Bergeon of sulphuretted hydrogen, they have not, I think, been proven in other hands to possess the notable efficacy which their originators claimed.

The general indications in cases of laryngeal phthisis are practically the same as those to be observed in the management of an ordinary case of pulmonary involvement, and therefore need not be fully discussed in this connection. Of course, in connection with our efforts to control the local process by topical agents, we avail ourselves at the same time of the beneficial effects which can be obtained by the administration of cod-liver oil and iron, general tonics, creosote, and other internal remedies which clinical experience teaches us to possess a certain value in tubercular disease. The administration of creosote in these cases, I think, should be specially urged, for while we do not fully indorse Robinson and others who advise that it should be given in doses of from gr. xl. to l. daily, I have seen most excellent results follow the use of gr. i. to ij. given three times daily. According to my observations large doses of

creosote are not well tolerated but are liable to cause disturbance of the stomach, and more especially of the intestines, hence any increase of the dose should be made carefully.

As regards the enforcement of certain hygienic rules, such as the regulation of the clothing, proper ventilation in living and sleeping-rooms, the use of the bath and matters of this sort, together with a change of climate where available, we must proceed according to the same rules, of course, which govern us in giving instructions in the case of pulmonary disease.

Perhaps no symptom in laryngeal phthisis is the cause of more suffering and distress than the dysphagia; so great is this, indeed, that it often becomes a serious problem to enable the patient to take an amount of food sufficient for ordinary nourishment. The assistance which we derive in these cases from the use of cocaine has already been alluded to, and in controlling painful symptoms this drug is fully as important, if not more so, than opium. Wolfenden has made rather an ingenious suggestion in observing that one of his own patients, in whom deglutition was usually painful, found that he could take fluids with considerable ease by stretching himself prone upon his stomach with the head lower than the feet, and sucking them through a tube. Delavan has devised an apparatus for alimentation in this disease, which consists of a flexible catheter attached to a pumping apparatus, by which the food is forced into the stomach. The same purpose in many instances could be accomplished by an ordinary nasal douche or by a fountain syringe with a catheter attached.

Lupus of the Larynx.

This curious affection was in the earlier literature regarded as a disease which, under the names of lupus exedens and lupus non-exedens, confined its ravages entirely to the integument, excepting in those somewhat rare instances in which by continuity of tissue the nasal passages became involved.

The possibility of its primarily invading the air tract seems to have been entirely overlooked, those cases in which it occurred being regarded, especially by French writers, as instances of strumous disease. In 1835, however, we find Rayer in his treatise on diseases of the skin suggesting that the morbid process which he observed occurring in the fauces of patients suffering from cutaneous lupus might be the same. This view was adopted by Hamilton, Cazenave, Alibert, and others in observations made during the following twenty years. As late as 1874 we find Isambert, Bazin, Landrieux, and others

teaching that those faucial ulcerations which we now recognize as lupus were manifestations of the strumous habit. This term "struma" is one which at the present writing we can probably safely eliminate from medical literature, in that it describes no definite morbid condition. The large majority of cases probably which have been recorded as instances of strumous ulceration were undoubtedly cases of lupus, although many perhaps have been either congenital or acquired syphilis. Lupus, then, is a term which we use to designate a specific morbid condition which in a large majority of instances arises primarily in the skin, but may also attack, primarily or secondarily, the mucous membrane of the nasal passages, the fauces, or the larynx.

Etiology.

No actively exciting cause can be assigned for the disease. As a matter of clinical observation, in many cases where it has arisen in the integument the cause has been directly traced to some injury, its subsequent development in the mucous membrane being by continuity of tissue; but in those rare instances where it attacks the mucous membrane primarily it seems to develop spontaneously.

Heredity does not seem to influence the disease, although unquestionably a constitutional tendency, which bears a certain relation to the tuberculous diathesis, plays an important part in its causation. It is a generally accepted teaching at the present day that the special morbid lesion which constitutes lupus is due to a tuberculous deposit which is identical with that which is present in tuberculous disease of other organs. In one case, however, the morbid process is an active one, while in lupus the morbid process is essentially a chronic or latent one, so latent as practically to constitute a local lesion rather than a systemic invasion.

I have collated 82 cases of lupus which invaded the air tract, including those which occurred in my own practice. Of these 51 were females, while 21 were males, the sex not being reported in the other cases. That the larger number of cases should have occurred in females is a curious fact for which it is impossible to suggest any explanation, since the pathology of the disease naturally suggests certain clinical relations between lupus and phthisis, and in the latter disease by far the larger number of cases occurs among males. Of the above 82 cases, 2 occurred in the first decade of life, 24 in the second, 26 in the third, 9 in the fourth, 6 in the fifth, 1 in the sixth, and 1 in the seventh. In 13 the age was not reported. We thus find that the favorite period of life for its occurrence is between the ages of ten and thirty. It is probable that in many of the above cases the

time of development was even earlier than the age given in the reports. This would suggest to us that the development occurred at an earlier period of life. In the majority of instances, therefore, it probably originates in the second decade of life. This, of course, is entirely with reference to the disease as met with in the mucous membrane. Cutaneous lupus occurs as a rule at a somewhat earlier period. Thus in a number of cases reported by Chiari and Rhiel the cutaneous lupus in six instances developed between the ages of two and five.

As before stated, in the majority of cases the disease commences in the integument, and subsequently, at periods varying from one to ten or twenty years, invades either the pharynx or larynx by the development of new centres of infection, or the nasal cavity by continuity of tissue. A number of cases of primary laryngeal lupus have been reported, and four such instances have come under my own observation. As evidencing the slow progress of the affection, in one patient, a man twenty-eight years of age, the disease in three years and a half had destroyed two-thirds of the epiglottis and infiltrated the aryepiglottic folds and commissure (see Fig. 70). The skin was not involved nor was any other portion of the air tract.

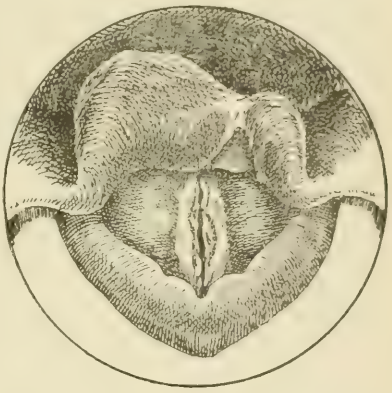


Fig. 70.—Lupus of the Larynx (Author's Case).

When the disease extends to the air tract and fauces it first attacks, in most instances, the soft palate and pharynx and subsequently invades the laryngeal tissues. Of the 82 cases collated above of lupus of the air tract, the pharynx, larynx, and skin were involved in 31, the larynx and skin in 19, the pharynx and larynx in 14, the pharynx and skin in 5, and the larynx alone in 12. In most of these instances the order of development was as given above—viz., first in the skin, subsequently it involved the pharynx, and still later the larynx. In one of Chiari and Rhiel's cases, a male aged twenty-one years, the cutaneous lupus developed during childhood and subsequently invaded the larynx, while the pharyngeal invasion occurred at a still later period. Another case reported by the same observers, in a girl of fourteen, was somewhat unique, in that the primary invasion occurred in the fauces at the junction of the hard and soft palates, while the larynx became involved later, apparently from a new centre of development.

The close relationship between lupus and phthisis seems clearly established on pathological grounds and this is reinforced by clinical investigation, for while the two affections seem to pursue a totally different course, their coincident occurrence in the same individual in such a large number of cases seems to establish this clinical relation as far as cutaneous lupus is concerned. When we come to consider lupus of the air tract, however, this teaching is not fully borne out, since I find but a single case, viz., that of Thoma, in which lupus of the throat occurred in an individual showing evidence of tuberculous deposit in other regions. Bloch, however, in a compilation of 144 cases of cutaneous lupus found 114 instances in which a tuberculous process in other organs either preceded or followed the development of the cutaneous affection. On the other hand we find Raudnitz, in a compilation of 209 cases of lupus of the skin, reported but 2 instances of a developed tuberculous disease in other organs, although a family history of tuberculosis was found in 21 of these cases, while an hereditary taint was probable in 9 others. Besnier found in 38 cases 8 in which there was tuberculous disease of other organs. To recapitulate, we thus find in Bloch's statistics a tuberculous taint in over 75 per cent. of his cases; in Raudnitz in 9 per cent.; Besnier in 20 per cent. These statistics would seem to conflict somewhat. We can only conclude that Bloch must have accepted as evidence of tuberculous disease, the existence of enlarged lymphatic glands, caries, and other manifestations which are usually grouped under the indefinite term "struma."

We find, therefore, this relation between lupus of the skin and phthisis to be clearly established from both a clinical and a pathological point of view. And yet when we come to the consideration of lupus of the air tract, we are scarcely justified in regarding tuberculous disease of the lungs or of other organs as standing in any but the most remote causative relation.

Pathology.

The assertion made by Neisser and Friedlander in 1881 that lupus constituted a true tuberculous process was in the following year verified by Koch in the announcement that he had discovered the presence of the tubercle bacillus in lupus nodules—a view which was still further confirmed and practically substantiated by the experiments of Schuller, Koch, and others, who produced tuberculous disease in the lower animals by the inoculation of lupus tissue, while Koch, going still further, produced pure cultures of tubercle bacilli from tissues invaded by lupus.

The most common point of invasion in the larynx is the epiglottis, the next in point of frequency being the ventricular bands and arytenoid commissure and then the vocal cords. After the primary invasion it extends by an exceedingly slow process of development to the neighboring structures without reference to anatomical boundaries. At the onset the disease seems to be one purely of infiltration, producing that peculiar nodular thickening of the parts by which their contour is destroyed. When the epiglottis is involved it becomes greatly thickened and markedly irregular in outline. It usually invades one side and the process extends laterally, showing no tendency to symmetrical development. Very soon after the infiltration occurs there sets in a process of ulceration which results in a notable loss of tissue. The ulcerative process which characterizes lupus is a peculiar one and differs in a marked degree from any other form of ulcerative action. There is an enormous thickening of the part from the primary and progressive infiltration, together with a slowly progressive recession or fading away of tissue, this without any of the usual concomitants of ulcerative action; the secretion is exceedingly limited, there is no pus discharge, no cell proliferation, no detritus, no necrotic tissue, and on close inspection it is difficult even to discover any ulcerative process. The loss of tissue is recognized, but no evidence of progressive action such as we usually see in ulcers. This may be accounted for, perhaps, by the fact that the process is an exceedingly chronic one, months and even years elapsing before any very great destruction is observable. The ultimate result, of course, is an extensive loss of tissue.

The essential pathological changes which occur consist of a deposit in the tissues or of their infiltration with small round corpuscles of granulation tissue, which may embed themselves between the bands of connective tissue and the glands and which show a tendency to follow the course of the blood-vessels. This infiltration, instead of occurring in a diffuse form, shows a disposition to gather itself into small masses, giving rise to the so-called lupus nodules. An examination of these nodules in many cases will reveal the presence of giant cells. Apparently we have to do, then, merely with a number of independent centres of inflammatory action which clearly indicate a morbid process due to the entrance of some specific virus; but in addition to this we notice the presence of the giant corpuscle which we know may occur in either tuberculosis, syphilis, or scrofula. The true significance, however, of these corpuscles in lupus is shown by the pathological investigations of Neisser, Schuller, and Koch, who demonstrate them to be part of a true tuberculous lesion.

Symptomatology.

An invasion of the larynx by lupus usually occurs in an exceedingly insidious manner. The parts are not rendered sensitive, there is no pain, there is no excessive secretion, there is no interference with deglutition as a rule, even where the epiglottis is invaded, although there may be a slight sense of irritation and possibly a cough. If the vocal cords are involved the voice may be affected and rendered hoarse, or complete aphonia may result from an invasion of the arytenoid commissure by which closure of the glottis is interfered with. If the disease has so far extended as to interfere with the free passage of air, dyspnœa, of course, sets in and may become an exceedingly distressing symptom. Practically, then, we find that interference with function is a far more prominent accompaniment of lupus of the larynx than any subjective symptoms to which the process gives rise. If there is much loss of tissue in the epiglottis we may have the additional symptom of the entrance of food or fluids into the larynx during the act of deglutition.

Diagnosis.

The epiglottis is usually the first structure to be attacked, after which the disease extends to the aryepiglottic folds and ventricular bands, causing thickening of tissue, distortion of contour, and impairment of function. The tissues are apparently slowly destroyed by a process of erosion involving both the mucous membrane and fibrocartilage of the epiglottis, but there is neither hypersecretion nor a distinctly ulcerated surface. The commissure and hyaline cartilages are affected in the same manner, but perichondritis with necrosis, such as occurs in tubercular disease, is, I believe, never occasioned. The subjective symptoms of the disease being of such an indefinite character, our diagnosis will mainly depend upon a laryngoscopic examination. Owing to the fact that the mucous membrane of the larynx is much more delicate in structure than that of the pharynx, the morbid process assumes a notably different aspect. In place of the very pronounced nodular appearance which lupus of the pharynx assumes with the extensive distortion of the parts, we find in the larynx that the prominent feature is loss of tissue, with no apparent effort at repair. At the central portion of the disease process we find an appearance of ulceration, but this has not the usual evidences of rapid waste of tissue, as commonly seen in ulcerative action. There is no apparent secretion. About the edges of the ulcer, and extending into the mucous membrane around it, small, pale-grayish, rounded

nodules are seen which cause a notable thickening of the part, whereby the normal contour is seriously distorted. We easily exclude syphilis, as in the latter disease there is marked hyperæmia, with notable evidence of rapid destruction of the part in the large secretion of pus mingled with necrotic tissue, while in lupus of the larynx the tissues are notably pale and exsanguinated, and there is no pus formation. In tuberculosis we also have a notably pale and exsanguinated appearance of the membrane, but the infiltration is more marked and the ulcerative action is pronounced, as shown by the large amount of thick, ropy muco-pus which usually covers the face of this form of ulcer. Moreover, a tubercular ulcer is exquisitely sensitive, and painful deglutition is a prominent symptom, in this differing essentially from lupus. These two diseases are practically the only ones with which lupus is liable to be confused, as in carcinoma and sarcoma the presence of the neoplasm should be easily recognized. Of course, when there is any question as regards diagnosis in a given case, a small mass can easily be removed by the snare or forceps and subjected to a microscopic examination.

Prognosis.

The disease in this situation is essentially chronic, as it is in the parts above. Laryngeal stenosis causing death from suffocation is the only source of danger, and is of course easily averted by tracheotomy.

So far as I know, there has been no case of spontaneous cure, but appropriate treatment at an early stage has often arrested the disease. The danger of stenosis would appear to be manifested within the first two or three years, after which time the disease seems to remain at a standstill, so far as this danger is concerned, rendering the question of prognosis somewhat difficult.

Treatment.

For convenience we divide the therapeutic measures to which we resort in the management of this disease into topical applications, the use of the curette, excision, and internal medication.

Topical Applications.—The various local remedies which have been used with more or less success are nitrate of silver, tincture of iodine, and lactic acid.

Bowen reports a case of faucial lupus cured by the application of a 480-grain solution of nitrate of silver. Ziemssen reports a case of laryngeal lupus cured by the solid stick of nitrate of silver. Hasland also reports an instance of faucial lupus in which the pharyngeal

geal disease was cured by the use of the solid stick of nitrate of silver, although the laryngeal affection progressed and subsequently demanded tracheotomy, after which the laryngeal invasion was arrested and cured by the same measure.

The use of lactic acid is naturally suggested by the success which has attended its use in laryngitis and tuberculosis of the larynx. I find, however, no records of cure by this agent, although it is warmly recommended by Ramón de la Sota.

Oberstuschen recommends very highly the use of the galvanocautery. This measure is one, however, which I think should be used with a great deal of caution in a laryngeal invasion of lupus.

Moure advises the use of chromic acid fused on the end of a small probe. This application is one which I am sure is to be highly commended and certainly in one case under my own care, in which the 480-grain solution of nitrate of silver failed to accomplish the desired end, the chromic acid produced very rapid improvement.

The Curette.—The admirable results obtained in the arrest of cutaneous lupus by this instrument naturally suggest its use in the air passages. Whereas this measure is easily available in the pharynx, I do not think that many cases of laryngeal lupus present themselves in which it can be used with success. Undoubtedly in certain cases, where the epiglottis is invaded, it can and should be employed. Its use should be followed by the application of nitrate of silver or chromic acid.

Excision.—This process is easily available in the early stage in many cases which first appear in the fauces, as on the soft palate or uvula, but in the larynx it presents difficulties which are not easily surmounted. Garre, however, reports an instance of a woman, aged twenty-eight, in whom the base of the tongue, epiglottis, aryepiglottic folds, and arytenoids were all involved. The disease had lasted a year. Local applications of lactic acid and iodoform failing to give relief, an excision of the epiglottis, and finally subhyoid pharyngotomy were done, and all the morbid tissue was excised. The procedure seems a somewhat daring one, but was fully warranted by its success in that there was no return of the disease at the end of five months.

Internal Medication.—In connection with topical applications resort should be had in all cases to such internal remedies as experience has taught afford the best results. These are cod-liver oil, arsenic, and iron. It is, of course, a somewhat nice clinical point to determine in those cases which have been cured how far local measures and how far constitutional remedies have contributed to the result. A careful reading of reported cases, however, carries the conviction that

general remedies have played an exceedingly important part in controlling the morbid process. In a case reported by Ramón de la Sota a cure seems to have been accomplished by the internal administration of arseniate of sodium alone. Whether in this form or in that of Fowler's solution, arsenic seems to possess somewhat of a specific action in the control of lupus, and our full duty is not done in any given case without fully testing its efficacy. Its action seems to be aided by the coincident administration of cod-liver oil and general tonics.

NEUROSES OF THE LARYNX.

A number of classifications of the laryngeal neuroses have been made by different writers, based variously upon anatomical, clinical, or pathological distinctions; the ordinary method being to make an anatomical classification as has been done by Cohen, Ziemssen, and Mackenzie in his earlier writings; in his latest work, however, Mackenzie's classification is based upon pathological distinctions, describing the various diseases according as they are dependent upon morbid conditions, respectively, of the medulla, spinal accessory, the pneumogastric, the superior laryngeal and the inferior or recurrent nerves. This classification is also adopted by Moure. Now we have certain rare diseases in the larynx which may be dependent upon an injury in the course of either the recurrent laryngeal nerve or the spinal accessory, or even as high as the medulla; thus pathologically the diseased condition is essentially different, while the clinical manifestation in the larynx is practically the same, hence it seems wiser to base this classification of the laryngeal neuroses on their clinical manifestions. In this way we shall consider—

1. Sensory neuroses, under which head we consider, hyperæsthesia, anæsthesia, paræsthesia, and general neuralgia;

2. Paralysis involving the nerve trunk or nerve centre, as follows: Superior laryngeal paralysis, inferior or recurrent laryngeal paralysis, bilateral paralysis of the abductors, and unilateral paralysis of the abductors;

3. Paralysis of individual muscles; these are: Unilateral paralysis of tension, bilateral paralysis of tension, unilateral paralysis of the adductors, bilateral paralysis of the adductors, and paralysis of the arytenoideus muscle;

4. Functional neuroses, the only affection coming under this head being hysterical aphonia;

5. Spasm of the glottis; this disease differs in such marked manner according as it develops in child life or in adult life, that it seems

wiser that we should consider it under the two heads of spasm of the glottis in children, and spasm of the glottis in adults;

6. Incoordination of the laryngeal muscles, including chorea of the larynx, aphonia and dysphonia spastica, and laryngeal vertigo.

Hyperæsthesia.

We generally say that the mucous membrane of the larynx is especially sensitive, or hyperæsthetic, in certain disease processes. This is notably true of tuberculous disease and in some instances of carcinoma. It is also to a certain extent observable in connection with acute laryngitis, acute pharyngitis, and in a less measure in chronic inflammation of the larynx and of the pharynx, especially where the latter results from the excessive use of alcohol. The reflex sensibility of the larynx varies notably in different individuals according to temperament, and is usually to be regarded as simply symptomatic or indicative of the general habit.

Anæsthesia.

Diminution or abolition of sensation of the larynx is necessarily the result of some involvement of the superior laryngeal nerve, though occasionally we meet with diminished sensibility in a chronic inflammatory process of long standing. It may also occur in syphilis and is one of the sequelæ of diphtheria. It is usually present in general paresis and in cases of tumors of the brain and in the early stages of tabes dorsalis and other affections of the medulla.

Paræsthesia.

This is a term which we use to designate perverted sensations which are occasionally met with in the larynx or fauces, or, rather, conditions which we call perverted sensations—such as tingling, a sense of irritation, lancinating pains, a feeling as of a foreign body in the throat, what a patient sometimes describes as a "cottony" feeling in the throat, a constant disposition to swallow, or a feeling as of a foreign body impinging upon the parts. In a large majority of instances in which these apparently perverted sensations occur, a careful investigation with a good illumination will not infrequently reveal some organic lesion to account for the symptoms, such as hypertrophied lingual tonsil, enlarged lymphatics in the pharynx, or cheesy masses in the tonsil. Occasionally a tickling or sense of irritation producing cough is referred directly to the larynx, whereas the real trouble on investigation will be found to lie in the passages above.

In individuals in whom the general health is impaired by anæmia, chlorosis, or phthisis, and especially in cases of neurasthenia, these perverted sensations become the source of constant discomfort. During the illness of General Grant and that of the German Emperor it was very curious to observe how constantly individuals exaggerated the importance of trivial symptoms referable to the throat, in many cases these being purely imaginary; while in other instances some trivial symptom, by brooding and worry, was exaggerated and intensified to such an extent that it became a source of notable discomfort and even pain. So closely related are the various parts of the upper air tract both by innervation and function that it is usually by no means easy for a patient nicely to locate the source of a sensation, which would serve to emphasize the teaching that a symptom referable to any one part of the air tract should demand a careful inspection of the whole area to determine and eliminate such morbid conditions as are discoverable in any portion.

Neuralgia.

A subjective symptom of pain referable to the larynx is by no means uncommon; in the majority of cases it has its source directly in some morbid condition of an organic character, either in the larynx itself or in the adjacent passages. It may be due to general conditions, such as anæmia, rheumatism, or gout, but it is especially prominent in the ulcerative processes of phthisis and carcinoma. It is not characteristic of either lupus or syphilis. The pain which occurs in connection with acute inflammatory troubles and which is especially prominent in perichondritis and certain injuries is, of course, purely symptomatic and need not be considered in this connection. Chapman has reported a number of instances in which the pain of a somewhat severe character, which was referred to the larynx by the individuals, was the result of a malarial poisoning; although there was a certain amount of tenderness upon pressure, the larynx revealed nothing abnormal. A cure was effected by the administration of iron and quinine. Schnitzler has observed a case which possesses a somewhat unique interest in that the pain seems to have been almost purely neuralgic in character. The neuralgia came on immediately after an attack of angina, and caused such intense suffering that the patient was almost driven to suicide. A cure was effected at the end of a month by a daily application to the larynx of a solution of chloroform and morphine. A somewhat similar case came under my own observation some years since which possesses so many points of interest that it is briefly related: I was summoned to a neighbor-

ing city with the request to bring instruments for the performance of tracheotomy in a very urgent case. I found a gentleman aged thirty-five, suffering from an intense pain in the larynx, with a feeling of constriction about the throat, which, while not causing any dyspnœa whatever, gave rise to a subjective sensation of imminent suffocation. The larynx was examined with difficulty but found to be perfectly healthy and the movements of the glottis normal. The patient was suffering from an acute naso-pharyngitis at the time, while the prominent neuralgic symptoms were explained by the fact that he was of an intensely nervous habit. I prescribed aconitine in gr. $\frac{1}{800}$ doses, to be administered every two hours until the characteristic symptom of tingling in the fauces was produced. The patient was completely relieved and had had no recurrence when seen three years later. In this case the laryngeal pain seems to have been purely neuralgic in character, being paroxysmal, while in Schnitzler's case the paroxysmal element, so characteristic of pure neuralgia, was absent.

The first prominent indication for the treatment of any of these sensory neuroses of the larynx consists in the removal of such morbid conditions as may be discovered in the air tract which may act as contributing causes. In most instances this will prove sufficient. In those cases in which the impairment constitutes an independent neurosis, our management of the case will be based upon those general rules which govern us in the correction of a neuralgic tendency, which in a general way are embraced in the judicious use of the cold bath, sea-bathing, exercise, friction to the skin, massage, or possibly change of climate, together with the internal administration of some preparation of iron, arsenic, or zinc, and such other drugs as may be indicated for the correction of general systemic conditions.

Paralysis of the Superior Laryngeal Nerve.

While the superior laryngeal nerve supplies sensation to the mucous membrane lining the larynx, it also contains a few motor fibres which give innervation to the crico-thyroid muscle and in part to the arytenoideus. A complete paralysis of this nerve would, therefore, result in an abolition of sensation of the whole of the mucous membrane lining the larynx, together with a motor paralysis of these two muscles, giving rise to a diminution of tension of the cords and imperfect approximation of the arytenoid cartilages. This would occur on one or both sides according as one or both nerves were involved. An interesting case was observed by Heymann of bilateral paralysis of these nerves which occurred in connection with paralysis of the constrictor muscles of the pharynx, rendering deglutition

impossible. An investigation of the case by means of a probe introduced into the larynx showed a complete abolition of sensation in its membrane, while at the same time the laryngoscope revealed the cords in a notably relaxed condition. On attempted phonation the arytenoids were rotated inwards easily, so that the vocal processes were in contact. As the result of the relaxed condition of the cords the glottis formed an ellipse anterior to this point, while posteriorly, on account of the paralysis of the arytenoideus muscle, there was a triangular opening extending back to the arytenoid commissure. This is shown in Fig. 71. The voice was weak and at times almost completely lost. Under the administration of general tonics and the local use of electricity, the patient ultimately recovered. No suggestion regarding the cause of the attack is made.

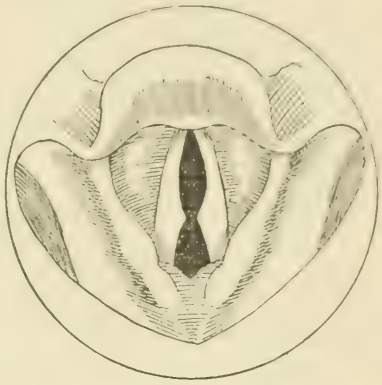


FIG. 71.—Bilateral Paralysis of the Superior Laryngeal Nerve.

Several instances have been observed in which the superior laryngeal nerve of one side has been cut during operations or in assaults or suicides, in which the result was simply relaxation of one cord as shown by a bulging above its fellow, in the act of phonation, although the arytenoid approximation was not interfered with, this muscle receiving sufficient innervation from the nerve of the opposite side.

A far more common involvement of the parts supplied by this muscle has been observed as one of the sequelæ of diphtheria, a prominent feature being the impairment of sensation in the laryngeal mucous membrane together with vocal weakness. The attack usually occurs in connection with impairment of motility of other muscles in this region, such as the muscles of deglutition. It has also been observed after typhoid fever, and may be a manifestation of hysteria.

The *diagnosis* of this rather rare but interesting form of paralysis will be based upon a careful study and analysis of the laryngeal movements as observed by the laryngoscopic mirror, in connection with proper tests of the sensation of the parts by the introduction of a probe.

I know of no lesion or condition which will produce the somewhat curious glottis which is observed where both the superior laryngeal nerves are paralyzed, the interesting point being that the vocal processes and arytenoid cartilages are rotated inwards in such a way that

the vocal processes are the only points which are in contact during phonation; an elliptical opening being observed in front and a triangular opening behind, as seen in the illustration. If, on the other hand, we have a unilateral paralysis, this triangular opening between the vocal processes is not seen, the contraction of the arytenoid muscle being accomplished through the innervation of the nerve of the opposite side; hence the laryngoscope shows, then, merely a relaxed condition of the cord, extending from the vocal process to its anterior insertion—a condition seen in paralysis of the crico-thyroid muscle (see Fig. 72). The impairment of sensation in the mucous membrane lining the larynx would indicate a lesion of the superior laryngeal nerve.

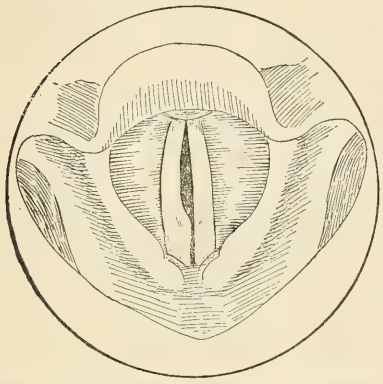


Fig. 72.—Unilateral Paralysis of the Superior Laryngeal Nerve.

These cases generally recover. If they follow diphtheria a complete recovery may be anticipated at the end of from about one to two months. In a case of section of the nerve reported by Creveling, complete restoration of the voice did not take place until the end of twelve months.

The *treatment* consists in the administration of general tonics and strychnine, in friction, massage, and local faradization, with other such measures, hygienic and otherwise, as may be indicated.

Recurrent Laryngeal Paralysis.

The recurrent or inferior laryngeal nerve supplies motor innervation to all the muscles of the larynx except the crico-thyroid. In consequence of the paralysis of this nerve, therefore, we have practically a complete abolition of motility in all the muscles on the side of the larynx involved; for although the crico-thyroid, receiving its innervation from the superior laryngeal, retains its motility, yet where all the other muscles of the larynx are in a state of paralysis it becomes practically impossible for this single muscle to exert any appreciable influence.

In the large majority of cases which come under observation, paralysis of the nerve is due to direct pressure upon its nerve trunk. Its course in the two sides of the body, as we know, is different; on the right side it winds around the right subclavian artery as it passes

up the side of the trachea, and on the left it winds around the arch of the aorta, where it seems to be exceedingly liable to become involved in aneurysmal dilatations of this vessel; hence our suspicion always in observing paralysis of the left side of the larynx, especially in a patient advanced in years, is that it is due to an aneurysm: yet, while aneurysm is a very common cause of the affection, it is probably a mistake to regard it as the most frequent cause.

Referring to the notes of my private cases of recurrent laryngeal paralysis I find fifteen cases in which the histories are complete. In four of these it occurred on the right side, and in eleven on the left. The ages varied from seventeen to sixty-eight. Eleven were males and four females. In but four instances was aneurysm discoverable. In one patient aged fifty-five the laryngeal paralysis was the only surviving symptom of what I regarded at the time as cerebral embolism, the paralysis occurring on the right side. The patient died a few months later with complete left hemiplegia. Embolism was the cause of the disease in another case of a patient aged thirty-three years, the paralysis being on the left side. In one case, a man aged twenty-five, it seemed to have been the result of an attack of diphtheria twenty-two years previously. In another case, a man aged sixty-four years, paralysis of the left side of the larynx occurred in connection with stricture of the œsophagus. In a boy aged seventeen, a left paralysis seemed to have followed an attack of whooping-cough. In two cases I have observed the left cord completely paralyzed and lying in the cadaveric position, during an attack of acute naso-pharyngitis with laryngitis. The condition disappeared completely at the end of a few days. One of these patients was a young lady aged nineteen and the other a man aged sixty-eight. Certainly in the latter case the paralysis foreboded no evil, in that the patient is still, after the lapse of years, in the enjoyment of perfect health. The observations were very curious ones, and I have no suggestions whatever to make in explanation.

The above cases afford, I think, a fair estimate as to the ordinary causes of the disease. That it occurs more frequently on the left than on the right side cannot be entirely explained by the fact of the nerve passing round the arch of the aorta, but is due in many cases to its being so much more exposed in its course than that on the right side, and nearer to the surface.

We learn from the above cases that paralysis of this nerve may result from a central lesion, a lesion in the trunk of the nerve, or from peripheral causes. Thus in two of the cases the paralysis was due to a central lesion, one occurring on the right side and the other on the left. Further proof that the paralysis may occur as the result of

a central lesion is shown by the remarkable series of cases observed by Jackson, Proust, Eisenlohr, and Sokaloff—in most of these instances the paralysis being unilateral, but in a number of cases being bilateral. In these cases the disease terminated fatally and the diagnosis was established by post-mortem examination. In every case the lesion was found in the medulla and involved the pyramids, the olivary bodies, the restiform bodies, and the floor of the fourth ventricle. The central lesion may be either hemorrhage, endarteritis, disseminated sclerosis, or the ascending sclerosis of locomotor ataxia. Clinical experience has shown that in the majority of instances the laryngeal lesion which results from, or occurs in connection with, locomotor ataxia is abductor paralysis of one or both sides, and yet a number of instances have been observed in which there was a paralysis of all the muscles supplied by the recurrent nerve.

The most frequent cause of paralysis of the recurrent nerve is pressure upon the nerve trunk in some portion of its course, and, as I have already observed, this occurs most frequently on the left side. This pressure is usually the result of aneurysm, enlarged lymphatic glands, or some other neoplasm in the course of the nerve, such as tumor of the mediastinum, cancer of the œsophagus, etc. Among the rare instances are those reported of this form of paralysis resulting from dilatation of the pulmonary artery. In another instance a pericardial effusion gave rise to pressure on both nerves. On the right side the nerve in its course runs very close to the apex of the lung; hence a pleuritic adhesion, especially that which occurs in connection with incipient phthisis, may cause pressure on this region and thereby paralysis of the nerve. Instances have been observed in which the paralysis was due to a serous effusion in the pleural cavity, and disappeared promptly on the resorption of the serum. In those cases in which the paralysis follows diphtheria, typhoid fever, and other of the exanthemata, we accept the teaching that the paralysis is due to blood poison causing a neuritis, involving either the nerve centres, the nerve trunks, or the terminal filaments. In two of my own cases, as well as in instances reported by others, a somewhat ephemeral paralysis of the recurrent nerve was observed in connection with acute inflammation of the faucial mucous membrane. The only suggestion here is that function was temporarily abolished, or that the nerve current was arrested by the involvement of the terminal filaments in the local morbid process. The occurrence of bilateral paralysis of the recurrent nerve should be accepted, in the very large majority of cases, as probably indicating a disease of the nerve centres, and yet we have recorded a number of rather unique cases, such as that of Bäumler, in which pericardial effusion pressed on both

nerves, and the still rarer instances reported by Ziemssen in which there was double paralysis, that on the left being due to aortic aneurysm, and that on the right being due to a subclavian aneurysm. Those curious cases in literature in which pressure on one recurrent nerve seems to have given rise to paralysis of both are difficult to explain, unless we accept the view that pressure on one nerve gives rise to such changes in the nerve centres as to lead subsequently to a paralysis of the opposite side.

Pathology.

Permanent interruption of the nerve current, from pressure on the trunk or destruction of the centre, is the cause of the degenerative changes in the nerve, which likewise extend to the muscles under the control of the nerve and bring about their atrophy from disuse.

Symptomatology.

In unilateral paralysis, so much increased effort is thrown upon the muscles of the unaffected side to bring the vocal cord into a proper position for speaking (the cord of the paralyzed side lying in the cadaveric position) that the mere effort at speaking becomes wearisome, and the voice grows weak, though not necessarily hoarse. After a few weeks the cord of the sound side becomes in a measure trained to the new position, and conversation is less difficult; but, the paralyzed cord being a trifle relaxed, its prolonged use becomes an effort, and the voice is limited in range. Still, on the whole, the improvement is marked, and after the condition has lasted some months no listener would be able to perceive any impairment of voice in ordinary conversation. In bilateral paralysis, however, there is complete loss of voice, because the cords are so widely separated that they cannot vibrate. When the patient attempts to speak in a labored whisper, the air escapes so rapidly through the glottis that he has to stop and take breath at the end of every two or three words.

Simple laryngeal paralysis being usually a symptom of some more serious affection, it is to the causative disease that such symptoms as dyspnœa, cough, etc., are to be attributed.

Diagnosis.

In bilateral paralysis, the laryngoscope will show the cords lying in a position midway between adduction and extreme abduction (see Fig. 73). In an adductor paralysis, which is the only affection likely to be confounded with this condition, the cords are more widely separated, and appear to be burying themselves in the lateral laryngeal

walls, while a somewhat concave appearance is imparted to them by the outward direction of the vocal processes. In the cadaveric position, on the other hand, there is a slight projection of the vocal process in the direct line between the arytenoid and the receding angle of the thyroid. When the paralysis is confined to one side, one cord will be seen lying in the cadaveric position, while the one of the healthy side not only moves as usual, but in adduction passes beyond the median line and goes over to meet its fellow. The arytenoid cartilage of the healthy side during this act passes a little in front of its companion cartilage (see Fig. 74).

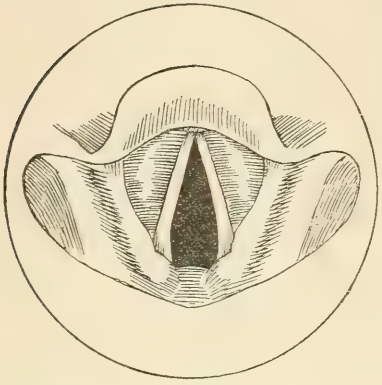


Fig. 73.—Cadaveric Position of the Cords, as in Bilateral Paralysis of the Recurrent Laryngeal Nerve.

This relative position of the two arytenoids during phonation will be one element in our diagnosis, the obliquity of the rima glottidis, which is deflected from before backward toward the paralyzed side, constituting a second. To detect this latter condition, arrange the mirror in the fauces in such a way that the centre of the crest of the epiglottis is brought in a direct line with the centre of the arytenoid commissure during inspiration (see Fig. 75). Then tell the

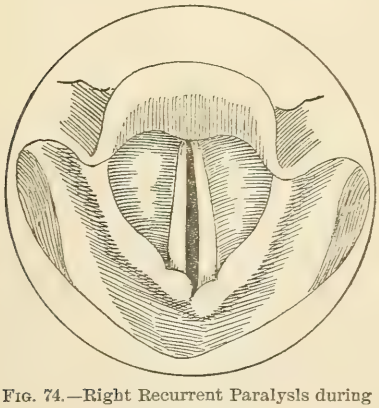


Fig. 74.—Right Recurrent Paralysis during Phonation.

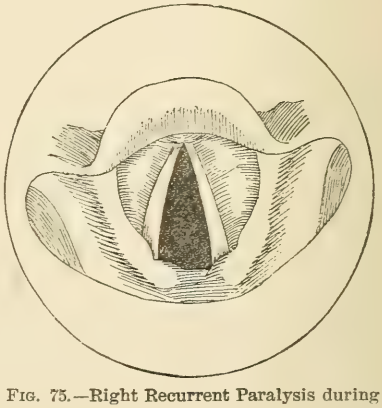


Fig. 75.—Right Recurrent Paralysis during Inspiration.

patient to phonate, and the narrowed rima glottidis will be seen in an oblique line while the cord of the paralyzed side lies motionless in the cadaveric position. An oblique epiglottis, such as we sometimes meet with, will give a deceptive laryngoscopic image, often resembling

a recurrent paralysis, in which case we shall have to adjust the mirror in the median line of the fauces, aligning it upon the soft palate and pharynx.

Not only must a diagnosis be made of the paralysis, but we must endeavor to ascertain its cause, to which end we shall have to examine carefully the thoracic organs by auscultation and percussion, the cervical region by inspection and palpation, and the nervous system by close questioning and other customary tests.

Prognosis.

Recovery may be expected in from three to six weeks if the disease result from peripheral neuritis, as in diphtheria, one of the exanthemata, or an attack of acute inflammation. Where, however, it is due to central lesion or to pressure on a nerve trunk, the result will depend upon the features of the more serious affection of which it is a symptom. If this be a curable disease, the functional restoration of the larynx will depend upon the duration of the laryngeal symptoms. The conductivity of a nerve may in rare instances be restored after paralysis has existed for more than a year, but usually after six to nine months the muscles supplied by the nerve are subject to such atrophic changes that their restoration is doubtful.

Treatment.

If the cause of the affection is an ordinary inflammatory process of the upper respiratory tract, we should endeavor to remedy the condition as rapidly as possible by means of local astringent applications. When it is a sequela of diphtheria or one of the exanthemata, general tonics and strychnine should be given. Where a diseased nerve trunk or centre is at fault, treatment will depend upon the cause of the paralysis. Unless this be ascertained and removed, the only use of electricity will be to maintain the integrity of the muscles until the cause is taken away. Neither can electricity be of benefit in the case of aneurysm or other incurable disease. But a tumor pressing on a nerve trunk may sometimes be removed by electrical stimulation, and where there is any hope of this it should be methodically and persistently applied. One pole of the battery may be placed upon the nape of the neck, and the other over the laryngeal region in front, or preferably into the larynx, and the various paralyzed muscles may then be stimulated. Cocaine anæsthesia will facilitate this procedure. The faradic is usually the more efficient current to use, but occasionally the continued current will show a better reaction.

Electricity is so rarely beneficial in cases of recurrent paralysis,

and does so often irritate and inflame the parts, and exhaust the strength of the patient, that it should never be used unless there is a very fair promise that it will be of decided benefit.

Bilateral Paralysis of the Abductor Muscles.

The respiratory function of the larynx, or the opening of the glottis by muscular movements, depends altogether upon the action of the posterior crico-arytenoid muscles, and is presided over by distinct nerve centres in the medulla.

Etiology.

Three theories have been held as to the causation of this disease, the first that it is due to a peripheral lesion, the second that it is caused by a morbid lesion in the continuity of the nerve, and the third that it is due to a central lesion. Mackenzie at first held that the cause of the disease was usually a cerebral lesion, but later apparently abandoned this idea and thought that the exposed situation of the abductor muscles on the posterior aspect of the larynx and their ceaseless activity rendered them especially liable to injury and that this liability might account for the disease. Smith, in the report of a case of this nature, thought that progressive weakening of the posticus muscles resulted in a species of prolapse of the posterior laryngeal walls, causing increasing dyspnœa. Gowers thought that pressure of the nerve trunk results in a paralysis first of the abductors and secondarily of the adductors, owing to the acute angle of the insertion of the former into the arytenoid cartilages, and the right angle of the insertion of the latter.

Krause appears to hold that the disease is caused by a spasmodic contraction in the laryngeal muscles, which may result from a morbid condition of the nerve centres, or depend upon a reflex irritation of the superior laryngeal nerve or a direct irritation of the recurrent nerve. I do not question that this spasmodic element may be present in some of the rarer and more obscure cases, but I cannot believe that it has any bearing on those which have been affected for a long period of time; for in spite of the explanations offered, I do not see how a tonic spasm affecting a given group of muscles can persist for years even, without resulting in degenerative changes which are to an extent uniform in all, for the abductor muscles are the only ones which undergo marked atrophic degeneration. As to a lesion in the continuity of the nerve, a number of authorities have held that pressure on the recurrent laryngeal nerve, or some other condition which might interrupt the nerve current, will explain the phenomena of abductor

paralysis. Semon from a long series of experiments deducts the inference that where the nerve current supplying all the laryngeal muscles is interfered with or checked by pressure exerted in the course of the nerve, the fibres supplying the abductor muscles are the first to succumb, or, in other words, these muscles are somewhat weaker than the other muscles of the larynx and are the first to yield to a defective innervation. Experimentation has, it is true, demonstrated that under electrical stimulation the adductor fibres show a greater vitality than the abductors, but I fail to see that this proves the proclivity theory. We might naturally expect that there would exist a difference in the response to electrical stimulation on the part of the phonatory and respiratory muscles of the larynx, since the first are only called into play voluntarily and in an intermittent manner, while the latter are active involuntarily and unceasingly from birth until death. A certain difference in innervation would therefore exist of necessity. I hold that the view that the disease is due to pressure on the recurrent nerve, and that this pressure so far discriminates between the nerve fibres as to destroy the conductivity of those fibres alone which are distributed to the abductor muscles, is quite untenable. But I do believe that clinical experience will prove the cause to consist in a morbid condition of the nerve centres, and still hold to my opinion expressed as follows in a former article upon the subject: "Reasoning from analogy, considering the peculiar character of the respiratory movements of the larynx in that they are purely involuntary and also reflex, that the opening of the glottis constituting the respiratory movement is an independent action separate from all the other movements which take place in the larynx as the result of muscular contractions, it is fair to conclude that this function is presided over by an independent ganglionic nerve centre, and that the disease in question consists in some degenerative change taking place in this portion of the nerve centres."

The clinical history of locomotor ataxia substantiates this view, showing that laryngeal crisis, anæsthesia of the lining membrane of the larynx, loss of reflex irritability, and in short, symptoms of paralysis of the abductor muscles are a frequent accompaniment of the disease.

Pathology.

An interesting feature of the disease is the fact that the abductor muscles themselves undergo degenerative changes with atrophy, purely as a result of the interrupted nerve current, and these changes are completed, probably, in from six to eight months.

Symptomatology.

Inspiratory dyspnœa, soon becoming spasmodic in character, is the chief symptom of the affection. At first mild in nature, the attacks become serious and occur with greater frequency, even two or three times a day. Expiration is not affected, nor is the voice impaired. During the intervals of attacks the patient breathes with ease.

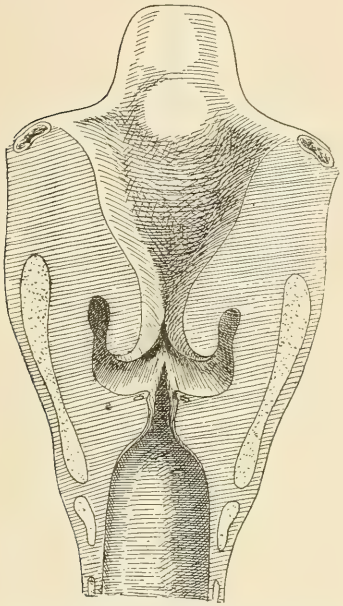


Fig. 76.—Transverse Section of the Larynx, Illustrating the Valve-Like Action of the Cords in Bilateral Paralysis of the Abductors.

A transverse section of the larynx, as seen in Fig. 76, shows that the upper surfaces of the vocal cords and adjacent tissues are hollowed in such a way as to present a valve-like orifice when closely approximated, something like that of the semilunar valves of the aorta. When the air is inspired it has a tendency to render the closure more complete, and this, I think, will in part explain the dyspnœic attacks. Aside from these attacks, the other symptoms will be those of the disease occasioning the laryngeal paralysis, such as a central nerve lesion, aneurysm of the aorta, tumor of the mediastinum, bronchocele, enlarged lymphatic glands, locomotor ataxia, etc.

Diagnosis.

A laryngoscopic examination will, during the act of inspiration, show the chink of the glottis narrowed and the cords in a position of parallelism (see Fig. 77), the space left being from one-eighth to one-tenth of an inch. In expiration the rima is opened by the current of air. During phonation the cords behave as in health.

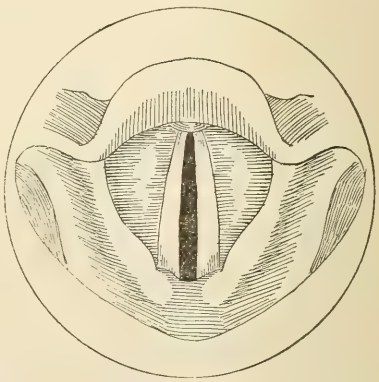


Fig. 77.—Bilateral Paralysis of the Abductor Muscles.

The determination of the source of the laryngeal paralysis of course constitutes the most important part of the diagnosis.

Prognosis.

While return of motility of the abductor muscles when they have been paralyzed for over nine months is not to be hoped for, the condition is not dangerous to life, unless indeed the disease causing it be a fatal one. Dyspnœa is of course a threatening danger when severe, but the tracheotomy tube may prolong life indefinitely, as witness Mackenzie's case, where the patient wore a tube for twenty-four years, and died of some other disease. The cases in which the disease develops rapidly seem to hold out the best hopes of recovery.

Treatment.

Should examination warrant the belief that the lesion is peripheral, any local condition found in the larynx must be removed, the paralyzed muscles stimulated by electricity, strychnine administered, massage tried, and any other measures resorted to which promise relief. If the paralysis is due to pressure on the nerve trunk or to a central lesion, treatment of the larynx will be worse than useless. In a few cases, antisyphilitic treatment has resulted in a cure.

Tracheotomy is indicated wherever remedial measures fail, and should be performed before the dyspnœa becomes so severe as to render sudden suffocation possible. Moreover, after the tracheal opening allows the laryngeal muscles to rest, the internal medication and local electrization will give better results than when dyspnœic paroxysms are constantly occurring.

Unilateral Paralysis of the Abductors.

I believe that this form of paralysis, which does not differ in any marked degree from the bilateral paralysis, is also usually due to a lesion of the nerve centres. The symptoms show some differences, owing to the involvement of one side only.

Instances in which pressure on the nerve trunk produced this disease have been reported by several observers. It may in rare cases be due to peripheral conditions, as acute processes in the larynx. Gout, rheumatism, lead-poisoning, typhoid fever, diphtheria, and the exanthemata have all been known to cause it.

The image seen in the laryngoscope is normal during phonation, but during inspiration the cord of the affected side is without motion, while the other is abducted in its normal arc. In paralysis of the recurrent nerve of one side the cord is found in the cadaveric position, but the rima glottidis runs obliquely from before backward, the arytenoid cartilage of the unaffected side being drawn in front of its

fellow, while in unilateral paralysis of the abductors the chink of the glottis during phonation is in perfect alignment from before backward.

So little marked are the symptoms connected with the larynx that the affection is very often unsuspected, and discovered quite by accident. Dyspnœic symptoms are sometimes present, but rarely become more serious than a shortness of breath on exertion, and are probably due to the disease causing the paralysis, such as bronchocele, aneurysm, or other tumor pressing on the nerve trunk.

There is a possibility of the paralysis of one side becoming bilateral. The treatment is the same as in the double variety, except that tracheotomy is not called for.

Paralysis of Individual Muscles.

By this we mean an affection of the laryngeal muscles not already considered, in which the lesion is the result of myopathic causes.

These cases are neither very frequent nor very important, excepting perhaps when the arytenoideus muscle is involved. As a rule, they occur as the result of a local inflammatory process, or they may be the result of overuse or straining of the voice. Sometimes the affection may be connected with anæmia, malaria, gout, or rheumatism, in which case I think that the general condition has usually started some local inflammatory process which has weakened the muscles and rendered them liable to loss of contractility from strain or overuse of the voice. "Hysterical aphonia" will be considered later on, as being a paretic rather than a paralytic affection, and not due to a local lesion.

All the so-called myopathic paralyses have probably the same etiology, but require a separate consideration in regard to their other features.

Unilateral Adductor Paralysis.

Some cases of this nature have been reported, but I have never seen one, and I hold with Lefferts that it would require great nicety of observation to draw a distinction between extreme abduction and the cadaveric position of the cord. I do not believe that it can occur from injury of the pneumogastric, as some have claimed, or from any known lesion of the central or peripheral nervous system. Its character must be purely myopathic; in this form it has been known to follow lead-poisoning, diphtheria, exposure to cold, etc.

Impairment of the voice, or aphonia, is the only symptom.

Upon examination, one or the other cord will be seen lying motionless in extreme abduction. During phonation the cord of the

unaffected side is brought around as far as it can be toward the immovable cord, its arytenoid coming in front of the other. It is difficult to diagnose between this condition and that of recurrent laryngeal paralysis, in which the cords are in the cadaveric position. The tensor muscles, however, are not affected in simple abductor paralysis.

Bilateral Adductor Paralysis.

Mackenzie has reported five cases of this disorder, which are the only ones of which I know. I think that they may have been hysterical in character, from the fact that there was noticeable movement in both cords.

Myopathic paralysis affecting the crico-arytenoid muscles might occur as the result of exposure to cold, lead-poisoning, diphtheria, scarlet fever, or other exanthem, and would occasion aphonia with phonatory waste.

Paralysis of the Internal Tensors.

Of all the forms of myopathic paralysis this is the most frequently occurring and most easily recognized. It is due to an impairment of contractility in the thyro-arytenoideus muscle, the situation of which renders it peculiarly liable to be included in inflammatory processes in this region. Thus it frequently occurs in connection with chronic laryngitis, but overstrain or overuse of the voice is usually the exciting cause. The voice is weakened and its range is diminished, notably in the higher notes. In ordinary conversation there may be no perceptible difference in the tone, but it may sink almost to a whisper if the muscular weakness is marked.

The affection may be unilateral, but is usually bilateral.

The *diagnosis* presents no difficulty with the aid of the laryngoscope. During phonation, instead of a narrow straight chink, we see an elliptical opening, due not to lateral separation, but to an upward inflation of the cords from the forcing of the air through the chink. The whole length of the cord is involved in the ellipse. Unilateral paralysis causes practically the same symptoms with reference to the voice, but the glottic opening presents a semi-ellipse (see Fig. 78). In paralysis of the crico-thyroid muscle there is im-

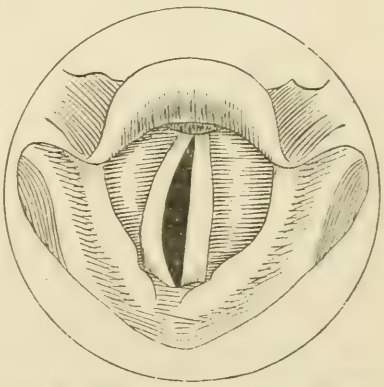


Fig. 78.—Paralysis of Left Internal Tensor.

pairment of phonation and an elliptical or semi-elliptical glottis, but the ellipse extends only from the receding angle of the thyroid cartilage to the vocal process. When the thyro-arytenoideus muscle is paralyzed, the vocal cord, which is practically its tendon, becomes narrowed and cord-like, whereas when intact it is flat and broad on its upper surface.

Paralysis of the Arytenoideus.

Chronic catarrhal processes in the larynx are very apt to involve this muscle. Incipient phthisis, catarrhal laryngitis, diphtheria, hysteria, and severe exposure have all been noted as the cause of the disease. The arytenoids, being unable to approximate perfectly, the voice becomes seriously affected or lost, because during phonation only that portion of the glottis is closed which extends from the thyroid angle to the vocal processes, which leaves a wide, triangular opening behind, as seen in Fig. 79. The air escapes through this opening during conversation in a most fatiguing degree.

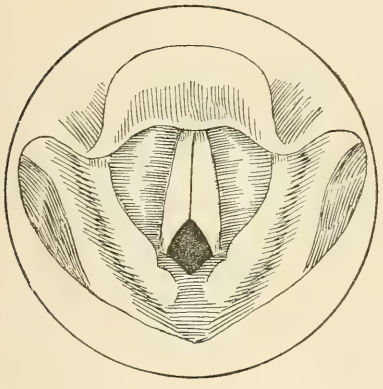


Fig. 79.—Paralysis of the Arytenoideus Muscle.

This condition may occur as the result of bilateral paralysis of the recurrent laryngeal nerve, but in this case would be accompanied by paralysis of the cricothyroid muscles, causing an elliptical opening of the glottis extending from the vocal processes to the thyroid angle.

Prognosis.

The question of the restoration of the voice is the only one of importance in myopathic paralysis and is less hopeful in proportion to the duration of the disease. Where it follows exposure, diphtheria, or one of the exanthemata the tendency is toward spontaneous recovery. The most obstinate of all these affections is paralysis of the internal tensors, because it is so difficult to secure absolute rest. In phthisis, myopathic paralysis would lead to the suspicion of some tuberculous process in the organ, but it should not be forgotten that the simple anæmia or impaired general nutrition accompanying phthisis predisposes to paralysis, which may then be overcome by appropriate measures.

Treatment.

Local lesions of the larynx must be removed, and rest of the parts secured by forbidding loud use of the voice or even by ordering the use of the whispered voice only—this especially in singers.

The electric current is our most efficacious means of treating the muscle, the faradic current perhaps giving the best results, but the continued current is to be applied if the other fail. Wherever practicable the application is to be made directly to the muscle itself. By the use of Ziemssen's apparatus one electrode is introduced into the larynx, and the other is applied over the larynx externally; this is with the single instrument. The double electrode is adapted for a more direct application of the current to the muscle within the laryngeal cavity. The treatment should be given daily, from ten to fifteen minutes at a time.

Strychnine given internally will aid the treatment, as will iron and general tonics, outdoor life, exercise, the cold bath if tolerated, followed by vigorous rubbing, and in fact all hygienic measures which are adapted to the case.

Hysterical Aphonia.

This is a purely functional disorder, characterized by complete aphonia and deficient muscular action, not a matter of the will power of the patient, and yet not a genuine paralysis. I would emphasize the point that although it has the appearance of being a counterfeit it is really not under the control of the patient.

Hysterical aphonia imitates only those paralyses which can be assumed by voluntary effort. Paralysis of the abductors is never met with, the abduction occurring only during inspiration which is a purely involuntary act. Unilateral paralysis of the vocal cords never occurs as a hysterical affection. Imperfect approximation of the cords constitutes the condition seen, and somewhat resembles the condition seen in bilateral paralysis of the recurrent nerve. The voice is lost, as the patient speaks in a whisper.

The hysterical affection may simulate subacute or chronic laryngitis, but the cords are normally movable in the latter disease, their thickened condition from swelling of the investing mucous membrane being the cause of the aphonia. It may also resemble the cases of aphonia due to mechanical interference with the closure of the cords by thickening over the arytenoid cartilages or commissure, but an examination will clearly show this condition. Bilateral paralysis of the recurrent laryngeal nerves is another affection which hysteria

simulates, but in this disorder all the muscles of the larynx are paralyzed, and the cords are absolutely motionless in the cadaveric positon—a position which cannot be assumed, for although the cords may be partially approximated, inspiration will widen the glottis, and motion can be seen to occur.

Hysterical paralysis is always bilateral, and there is always incomplete closure of the glottis. Beside the fact that a laryngeal image does not perfectly resemble any of the forms of genuine paralysis, the diagnosis will be assisted by the accompanying evidences of the hysterical temperament. Cough is present here, but absent in genuine paralysis of the adductors. The onset is apt to be very sudden, the patient perhaps awakening in the morning to find that she cannot speak.

Anæsthetics are the crucial test of the condition, the patient in the second stage of anæsthesia usually speaking with freedom. Women from fifteen to forty-five years of age, usually unmarried, are the ones most subject to this disorder.

Treatment.

The patient not being responsible for the condition, it should not be treated as unreal or dishonest, but, within bounds, as a real paralysis, and any existing exciting cause, as uterine disease, should be removed. In addition local treatment should be applied, to assist in convincing the patient that she can use her voice—not always an easy matter. To secure the patient's confidence is an essential point, and then she can be assured that the remedial measures to be adopted will at some definite time result in restoration of the voice. The preparations for tracheotomy in a case of Meschede's made such a profound impression that the paralysis was removed without performance of the operation.

Spasm of the Glottis.

Excluding the cases of so-called laryngismus stridulus which occur in catarrhal affections of the larynx, we reserve the term spasm for cases which are purely neurotic in character, and which consist in a sudden closure of the glottis that temporarily prevents the entrance of air.

Although some disturbance of the nervous system causes this disease, we must not suppose that any one lesion is affected in all cases. We will consider the affection under two headings, as the features which it presents differ greatly in infancy and in adult life.

Spasm of the Glottis in Children.

It is no more difficult to conceive of a laryngeal spasm of neuropathic origin in children than it is to understand that eclampsia or general convulsions are a condition of early life.

Etiology.

Rachitis is apparently the most frequent cause of this disorder. It has been suggested that this is due to softening in the occiput, but it seems more reasonable to infer that it is the result of impaired nutrition, as it also is liable to occur in children whose food and hygienic surroundings are deficient. Any condition which may set up reflex disturbances in young children, as prolonged crying, whooping-cough, exposure to cold, dentition, gastric or intestinal troubles, parasites, etc., may be an exciting cause, as may any condition causing increased sensitiveness of the upper respiratory tract. The pressure of enlarged bronchial or tracheal glands on the laryngeal nerves will also cause it; even the thymus gland may be so enlarged as to cause pressure.

The disease is usually met with between the first and the eighteenth month, and is more common among boys than girls.

Pathology.

The thyro-arytenoids, lateral crico-arytenoids, and the arytenoideus contract spasmodically and close the glottis, causing the paroxysm. Paralysis of the posterior crico-arytenoid muscles may also bring about an attack. The impulse causing the spasm may originate in the cerebrum, in the motor centre of the larynx, in the medulla, or may arise reflexly from some local trouble in the larynx, intestinal canal, or any portion of the body. Impairment of nutrition of the nerve centres from impoverished blood underlies these disturbances.

Symptomatology.

Without the slightest warning a child who has shown no symptom of laryngeal disturbance is seized with inspiratory dyspnœa, which may last from a few seconds to two or three minutes. This usually occurs at night, the child starting up in bed, giving labored crowing inspirations, struggling for breath and quickly becoming cyanotic. The glottis is narrowed during both inspiratory and expiratory movements, showing the spasmodic nature of the attack. Complete closure of the glottis sometimes checks respiration altogether for from ten to twenty seconds, being followed by several minutes of dyspnœa.

Several attacks may occur within twenty-four hours or they may recur only after an interval of several days, the seizures having a tendency to increase both in frequency and severity. This may persist for a few weeks and be followed by gradual improvement, or the child may die from suffocation during a specially severe attack. After recovery has apparently been established some slight exciting cause may bring on a paroxysm quite as menacing in its nature as any previous attack.

Besides the spasm, other evidences of irritation of the nerve centres are present, such as twitchings of the limbs, extension of the feet, clenching of the hands, and even diaphragmatic spasm.

The general condition causing the disease, together with the disturbed sleep and the tax on the nervous system, if it be of long duration, soon result in evidences of impaired nutrition.

Diagnosis.

Spasmodic attacks also attend laryngeal tumors, bilateral paralysis of the abductor muscles, and subglottic laryngitis. As the result of a new growth spasm is a rare symptom and mild in character, and apt to be masked by suffocative attacks. In bilateral paralysis of the abductors the affection is more chronic, the paroxysms longer and of less severity, and there is neither the rigidity of the glottis found in true spasm nor the convulsive movements of other parts of the body. Subglottic laryngitis causes nocturnal attacks of dyspnœa, but is usually accompanied by some fever, a hoarse, barking tracheal cough with expectoration, and impairment of voice, the dyspnœa occurring with inspiration only. The absence of these symptoms, and the presence of some rachitic or other blood condition, with convulsive symptoms evidencing the neurotic origin of the disorder, will form the basis of our diagnosis, and the age of the patient will render us further assistance.

Prognosis.

A large proportion of cases end in death, more perhaps in the case of boys than of girls. The gravity of the prognosis will depend upon the severity of the attack, the shortness of the interval between the spasms, and the extent to which the general health is affected.

Treatment.

To curtail the paroxysm we should admit plenty of fresh air, place the child in a semi-recumbent position, loosen the clothing, apply mustard plasters to the back of the neck, plunge the feet into hot water, and apply cold compresses to the head—all these measures

having for their aim to diminish blood pressure on the affected nerve centres. In a child of eighteen months gr. $\frac{1}{6}$ of morphine combined with gr. $\frac{1}{30}$ of atropine may be safely and profitably administered hypodermatically. If the paroxysms are prolonged, paregoric or musk, or castor may be given per rectum, but are not very promptly efficacious. Pressure on the pneumogastric nerves and on the carotid arteries has been recommended. Ammonia and chloroform inhalations might be of benefit were it not that respiration is, as a rule, completely arrested; but a few drops of chloroform injected in milk or water into the rectum might give good results. Emetics are probably of use only in such cases as depend upon a subglottic laryngitis. A prolonged paroxysm attended with unconsciousness and general convulsive movements will call for relief of the dyspnœa, either by intubation or tracheotomy.

Between attacks the system must be built up by hygienic measures, good ventilation, open air, nourishing food, proper clothing, and the daily cold bath. Cod-liver oil with the hypophosphites in connection with the syrup of the iodide of iron should be administered.

The act of nursing sometimes causes a spasm, necessitating feeding with a spoon. Considering this disease as closely allied to eclampsia, musk, castor, the bromide of potassium or sodium, or the two combined, chloral hydrate, and antipyrin, have all been tried, and may give good results. Physostigmine in half-drop doses of the fluid extract repeated every two to four hours until there are signs of muscular weakness, or curare in gr. $\frac{1}{30}$ doses may be given. The gums must be freely scarified if they are the source of irritation, and the bowels must be carefully watched, lest imperfect digestion be the cause.

Spasm of the Glottis in Adults.

Of a purely reflex nature, and depending upon an excitability of the nervous system rather than upon any morbid lesion of the nerve centres, spasm of the glottis in adults is not attended by danger to life.

It may be caused by the entrance of food, drink, or a foreign body into the larynx, by some irritating substance applied topically, and by the presence of movable tumors. Tubercular and syphilitic diseases give rise to it, probably because of the interference with deglutition caused by these affections, as a result of which food and drink may enter the larynx. Any disorder which interferes with this act is liable to cause the spasm, as pharyngeal paralysis, ulcerative processes in the pharynx or œsophagus, and swelling in any portion of the fauces. With the exception of these cases, the condition is probably to be regarded as a reflex phenomenon excited by some diseased

condition of the upper portion of the respiratory tract. The spasm, in rare cases, may arise from pressure on the efferent nerves; thus it has been known to occur from pressure of the recurrent nerve by a bronchocele, and from irritation of the plica nervi laryngei of Hyrtl by a hyperæmic condition of the pyriform sinus. Tetanic contraction of the muscles of the upper extremity has been given as a cause, and in the early stages of locomotor ataxia there is probably a spasmodic element in the laryngeal crisis. But although spasm of the glottis may be excited by a central lesion or by pressure of an efferent nerve, in the great majority of cases it is due to a reflex irritation originating in some part of the air tract, with perhaps a peculiar nervous excitability as a predisposing cause. It is, however, very doubtful whether hysteria could give rise to the affection, since I believe it to be a rule that it can only cause those disorders which can be perfectly simulated by purely voluntary effort.

Symptomatology.

The attack usually occurs at night, the patient being awakened by dyspnœa, with the labored and crowing inspiration, the struggle for breath, and rapidly supervening cyanosis characteristic of glottic spasm. This may last from five to twenty seconds and then subside, to be repeated the same night or perhaps not for a considerable period. There is no periodicity of the disease, as in children.

An attack at night would appear to be characteristic of the cases caused by reflex disturbance from a diseased condition of the air passages, while a morbid condition of the nerve centres or pressure on a nerve trunk would be more likely to cause an attack in the daytime, as is likewise the case with locomotor ataxia.

Diagnosis.

The affection itself is readily diagnosed, but its cause must be carefully sought for. If disease of the nerve centres and pressure on a nerve trunk have been eliminated, the seizures are probably reflex in character, and we may discover some abnormal condition of the respiratory passages, as atrophic or hypertrophic rhinitis, deflected septum, nasal polypi, pharyngeal adenoids, or other obstructive lesions of the nasal passages or in the naso-pharynx. Laryngeal crises, occurring in the early stages of tabes, are usually accompanied with or preceded by the loss of patellar reflex, the Argyll-Robertson pupil, and the peculiar ataxic gait.

In bilateral paralysis of the abductors which may cause laryngeal spasm, the absence of abductor action in the glottis will be easily detected by means of the laryngoscope.

Prognosis.

The disease is not fatal when it is of reflex origin, but causes great discomfort, and may necessitate tracheotomy. I have always found that the paroxysms yield readily to remedies directed to the cause of the affection.

Treatment.

To relieve the hypersensitive condition of the nervous system underlying this disease, we may administer bromide of potassium or sodium, in doses of from gr. x. to xv. three times a day, to be increased gr. v. daily until the effect is produced or signs of bromism appear. Cold baths, thin woollen underwear, exercise, and other hygienic measures should be prescribed.

Of course, any local condition liable to cause the attack, such as an atrophic rhinitis, must be remedied as far as possible, and much can be done to relieve the symptoms by this means, even where there is a morbid condition of the nerve trunk or centres. The irritability of the parts can be soothed by the application of a two-per-cent. solution of cocaine, inhalations of infusion of poppy, lupulin, conium, benzoin, or some other local sedative.

A tracheal cannula may in very rare instances have to be inserted temporarily to arrest the attacks until the cause can be sought and removed.

Laryngeal Incoordination.

A deficiency in the coordinate direction of the muscles of the larynx will cause a spasmodic contraction of the glottis, affecting expiration exclusively. Under this head we have, 1st, chorea of the larynx; 2d, dysphonia spastica; and 3d, laryngeal vertigo.

Chorea of the Larynx.

In this form there is momentary but complete closure of the glottis against expiration, followed by a sudden breaking through of the imprisoned air. The chief feature is a persistent, noisy, dry cough, resembling the bark of a dog. It is incessant except during sleep, and occurs every two or three minutes. Some cases show less regularity. The cough has apparently no relation to respiratory movements, and the tone of voice is unaffected. It occurs in girls more than in boys, and at about the age of puberty.

By means of the laryngoscope the spasmodic character of the glottic movements will be readily observed. During the intervals of

the attack the laryngeal movements are normal, but during the paroxysms the cords are driven together, as Lefferts expresses it, as if by some great external force, and after one or two seconds they are drawn back to the sides of the larynx by a similarly quick movement. A sudden expulsive effort appears to be excited in the expiratory muscles of the thorax by the glottic spasm, which opens the glottis and produces the loud, barking cough.

In some cases choreic movements in other parts of the body are met with.

Dysphonia Spastica.

In this affection the glottic spasm occurs only during the expiratory movement necessary for phonation. It is a disease of adult life, and is found more frequently in women than in men.

Impairment or loss of voice is the first symptom, soon followed by the peculiar spasms of the disorder, which occur whenever the patient attempts to talk. Sometimes the glottis is not completely closed, but there is so much tension that the voice becomes a falsetto. If the effort to phonate is persisted in, cyanosis may occur. By means of the laryngoscope we find normal vigorous movements of the larynx. An attempt to phonate will cause adduction of the cords in a perfectly natural manner, but the moment they approximate they contract spasmodically and close the phonatory glottis; one cord may overlap the other, and one arytenoid cartilage fall in front of its fellow.

Laryngeal Vertigo.

In this variety of spasm the glottic closure takes place when the lungs are filled with air and is of such a character that during its momentary persistence expiratory effort is unable to overcome it. The spasm is immediately followed by vertigo and loss of consciousness. An individual apparently in a healthy condition feels a slight tickling in the larynx and gives a cough, which is suddenly followed by obscurity of vision, dizziness, and complete unconsciousness which lasts a few minutes and then passes off completely, leaving no discomfort or weakness. Attacks may occur every few days, or at intervals of weeks or months. In mild cases there may be only the obscurity of vision and dizziness.

The arrest of expiration, and the unavailing attempt to force the air through the closed glottis, cause increased intra-thoracic pressure, interruption of the circulation, and lessened vigor of the cardiac contractions, with the result of a disturbance of the blood supply about the motor centres of the larynx in the medulla, and also in the

psychic centres of the cranial cavity. As a rule the larynx is normal, but sometimes there is a catarrhal laryngitis. The disease is rare. Its diagnosis presents no difficulty.

Prognosis.

Life is not threatened by any of these affections. Chorea and dysphonia spastica are essentially chronic, and often resist treatment for a long time. Laryngeal vertigo, however, seems to be amenable to treatment.

Treatment.

The neurotic condition which is the cause of these attacks should be dealt with first, and to this end may be given general tonics, such as barks and iron, cod-liver oil, and hypophosphites. Strychnine is contra-indicated. Sponge, shower, or tub baths of cold water have a stimulating and tonic effect: perhaps the shower and douche are the best means of securing the necessary amount of shock upon the system. Unless a proper reaction follow the baths they should be discontinued. General hygienic measures, relating to diet, clothing, exercise, and ventilation are of the utmost importance.

The predisposing cause of these attacks, as we have said, is to be looked for in a general systemic condition, but an active cause is very frequently to be found in some morbid process in the upper air passages, which must therefore be sought and removed.

Chorea of the Larynx.—In one case which came under my care, a complete cure was effected in three months' time by the internal administration of Fowler's solution of arsenic, and the correction of a nasal stenosis caused by hypertrophic rhinitis and deflected septum. An elongated uvula was removed in one of Lefferts' cases, giving great relief; the bromide of potassium, belladonna, and the faradic current completing the cure. Soothing applications give temporary relief even where there is no local lesion of the larynx or air tract, and with this intention we may use inhalations of conium, hyoscyamus, lupulin, papaver, or a local application of a solution of morphine or cocaine. Sometimes inhalations of hot steam are of value.

Bromide of potassium in full doses and the faradic current seem from all accounts to have given the best results, it being understood, of course, that they are prescribed in conjunction with hygienic measures.

Dysphonia Spastica.—The indications for treatment are the same as in chorea, with the addition of as complete rest to the organ as can be obtained, and the daily use of the constant current, the posi-

tive pole to be placed over the cervical vertebræ and the negative pole passed up and down the whole length of the spinal cord.

Laryngeal Vertigo.—In addition to the measures already referred to, bromides should be freely administered because of the marked neurotic element of the disease. A morbid condition of the upper air tract must be corrected, otherwise the bromides will be of no practical value.

FOREIGN BODIES IN THE AIR PASSAGES.

During the act of mastication, swallowing, or breathing, foreign bodies may enter the larynx or air passages below, oftentimes causing symptoms of grave import.

Etiology.

It would be difficult to give a complete list of the articles which may find their way into the larynx, since almost any substance that is in the mouth may, by a sudden impulse of laughter, or perhaps by sneezing, be drawn down. Pins, coins, particles of food, pebble stones, natural and artificial teeth, buttons, nutshells, lumbricoides, hydatids, etc., have all in turn caused the accident. The entrance of fluid during deglutition giving but temporary discomfort, except where there is tuberculosis or cancer of the larynx, we exclude it from the list. Some cases have been reported where the foreign body has come from below, as for instance that of a diseased bronchial gland which made its way into the bronchial tube by an ulcerative process and passed up into the larynx, or the occlusion of the trachea by cheesy matter from an ulcerating bronchial gland.

A cork inserted between the teeth during anæsthesia is known to have entered the air tract and to have caused death. Vomited matter, especially during anæsthesia or in the insensibility from intoxication paralysis, and also in new-born children, has been known to make its way into the air passages. In one case, while a young woman was eating, the epiglottis became impacted in the larynx. Leeches have been taken in with drinking-water and have entered the larynx. Children sometimes fall asleep with coins, buttons, toys, etc., in their mouth, a habit which occasionally necessitates a subsequent tracheotomy, as in the case where a toy engine passed into the air passage, causing dyspnœa of such alarming proportions that the operation had to be performed. By means of the laryngoscope its position was ascertained some months later, and by means of a thyrotomy it was extracted. The larynx of a goose was once removed from the larynx of a child after tracheotomy. False teeth

may fall into the larynx during sleep. In one instance of the kind the patient was unconscious of what had occurred, but the later development of a cough with dyspnœic symptoms and evidences of localized pneumonia led to a laryngoscopic examination, by which means the plate was found lying on the bifurcation of the trachea. It was removed by tracheotomy one hundred and thirteen days after the accident.

Foreign bodies are usually at once expelled from the pharynx, owing to its high degree of sensory and motor innervation, but during sleep this sensibility is diminished, and foreign bodies are then more apt to pass into the larynx and trachea.

A curious case is on record of an umbrella handle making its way into the left bronchus, causing pneumonia, purulent pleurisy, and death. A child five years of age swallowed a button hook, causing no symptoms for seven days following the accident, when vomiting, hoarseness, cough, and pain in the trachea set in, and ten days later the button hook was expelled during an attack of coughing.

The surfaces of the laryngeal walls are so irregular that one would expect a foreign body entering the air tract to lodge there. In 7 out of 15 cases collected by Durham they did so, going to the trachea in 5, the right bronchus in 2, and the left bronchus in 1 of the remaining cases. Out of 156 cases, however, Bourdillat found that the substance was arrested in the larynx only 35 times, in the trachea 80, in the right bronchus 26, and the left bronchus 15.

Symptomatology.

We have already mentioned instances where the entrance of the foreign substance was unperceived by the patient, no symptoms occurring until local inflammatory changes set in. The symptoms are sometimes light, but as a rule the accident is at once attended by the most distressing results, such as a sudden choking or gasping, and a feeling of suffocation. A spasm of the larynx occasions inspiratory dyspnœa—the patient rushes to the window for air, the eyes protrude, and the face becomes livid. Death may ensue, or the distress may subside, to be followed later by recurrent attacks of dyspnœa. A smooth, rounded, unirritating body may remain for a long time in the air passages without causing serious disturbance, as in the case of a pebble stone which was expelled ten years after its impaction, and in one of a penny which was removed from a child's larynx after having been there six years.

Usually, however, the presence of a foreign body is so irritating to the delicate structures of the air tract that inflammatory and ulcera-

tive processes are produced, which either are confined to the immediate vicinity of the foreign body or extend to the lungs and surrounding tissues, causing, according to their location, laryngitis, œdema of the larynx, inflammation or ulceration of the trachea or bronchi, emphysema, pneumonia, pleurisy, abscess of the lungs, and necrosis of the cartilages whether of the larynx or trachea. The continued presence of a foreign body in the larynx occasions hoarseness, aphonia, and recurrent or permanent laryngeal spasm, with cough and expectoration. In the trachea or bronchi it will give persistent cough, expectoration of a more or less purulent nature, and possibly tinged with blood, and dyspnœa.

A mistaken diagnosis of pulmonary phthisis is not infrequently made, owing to the fact that progressive loss of flesh, fever of a hectic type, night sweats, anorexia, and other marasmic symptoms are caused by the long-continued presence of a foreign body, with the ulcerative action to which it gives rise. Pneumonia, pleurisy, and other secondary affections may occur, and will occasion their special symptoms. Bronchitis assumes a peculiar intermittent type, distinguished by exacerbations with intervals of comparative freedom.

Local pain will usually serve to indicate the situation of the foreign body in the air passages; if impacted in the larynx that region will be the seat of pain; if in the respiratory tract below, the pain will be in the front wall of the chest over the location of the object, slightly radiating to the posterior wall.

Hemorrhage sometimes occurs, although it is not usually a marked symptom.

Diagnosis.

If the history of the case yield no information as to the accident, it will be somewhat difficult to detect the presence of a foreign body in the air passages, unless it be within the range of laryngoscopic vision.

On account of its anatomical position, the right bronchus is the more likely of the two to receive a foreign body; the pain induced will assist us in the diagnosis. Provided the tube is not entirely obstructed by the object, a harsh or sonorous râle will be perceived upon auscultation, and when it is occluded there will be absence of respiratory murmur in the portion of lung which it supplies. Cohen calls attention to the fact that occlusion of the right bronchus causes absence of respiratory murmur over the lower lobe of that side, while the same condition in the left bronchus affects the entire left lung.

Where the symptoms caused by the foreign body greatly resemble phthisis, and no history of the accident is obtainable, the diagnosis

is indeed obscure. If an examination of the sputa reveal no bacilli, and there is unilateral bronchitis with recurrent exacerbations attended by loss of flesh and hectic fever, and if the expectoration is of muco-pus tinged with blood, the suspicion of a foreign body may certainly be warranted.

Prognosis.

Life is seriously threatened by the entrance of a foreign body into the respiratory tract, either from immediate symptoms or from secondary morbid processes. Nature always attempts to get rid of a foreign body at once by cough and other reflex processes, and in the majority of cases accomplishes her purpose.

Out of 1,000 cases collated by Weiss, 72.5 per cent. recovered after operation, 77 per cent. without an operation. Of 139 cases ending fatally without operation, 84 died of asphyxia, 33 of pneumonia, 4 of exhaustion, 10 of abscess, 5 of laryngitis, and 3 of consumption. Of 271 cases reported by Durham, in which there was no operation, 57.5 per cent. recovered, 42.5 per cent. died; out of 435 cases 75.4 per cent. recovered after operation.

Of 85 cases not operated upon, which Gross reports, 56 recovered, 29 died. Among the 56 recoveries were included 3 in which emetics were administered and 4 in which inversion was practised, and of the 29 fatal cases 8 died after spontaneous expulsion of the foreign body. Out of 98 cases operated upon 83 recovered, 15 died.

Spontaneous expulsion has been known to take place in one day, and at the other extreme in sixty years, so that it would be difficult to set a limit upon its occurrence. It should, however, not be waited for, nor counted upon, but the object should be removed at once, with or without operation.

Treatment.

The character and location of the body must be ascertained as nearly as possible from the history of the case and the subjective symptoms, and by digital and laryngoscopic examination.

The first symptoms, such as choking and spasm, usually subside in a few minutes, and those which remain will indicate the gravity of the case. Dyspnœa so severe as to threaten suffocation necessitates immediate operative measures; with less urgent symptoms we should endeavor to obtain expulsion of the foreign body. Sternutatories and emetics are of doubtful value; it is better to have the patient take a slow, full inspiration and then violently force out the air, which procedure will be facilitated by violent blows upon the chest at the moment of expulsive effort. Inversion of the body is of great

assistance. The supine position is better than the prone, because if the foreign body lodge against the rima glottidis quick recovery of the erect position would avert spasm. With reference to smooth, round bodies, I think there is less danger than is usually supposed of impaction in the chink of the glottis during the attempt to secure ex-

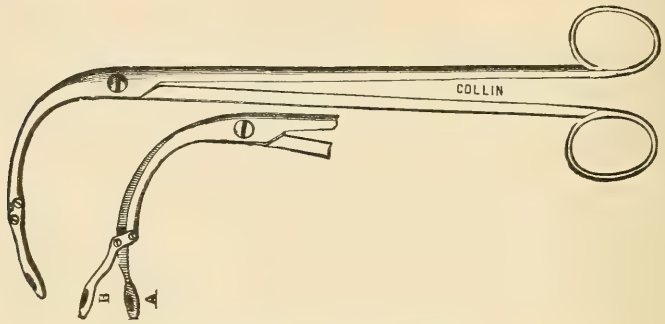


Fig. 80.—Cusco's Laryngeal Forceps.

pulsion by position; but when the body is of irregular shape and has sharp edges, there is ground for apprehension, and great care must be observed.

The next resource when the foreign body is in the larynx is the use of forceps and other extracting instruments, aided by the laryngoscopic mirror or the finger in the larynx. The Cusco forceps (see Fig. 80) combines a firmness of grasp with freedom of play of the blades, and is the best in the majority of cases. Figs. 81 and 82 show the position of a coin in the laryngeal ventricle in a case re-

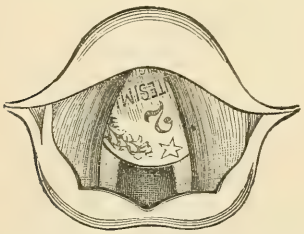


Fig. 81.—Coin in Laryngeal Ventricle (Grazzi).

Fig. 82.—Coin in Grasp of Forceps showing Method of Removal employed in Grazzi's case.

ported by Grazzi, in which the coin had to be grasped horizontally in order to extract it.

According to Gruening, preliminary tracheotomy is desirable where there is danger that the foreign substance may become dislodged and drop into the trachea during operation. To avoid tearing the tissues and perhaps permanently injuring the voice, it may be

advisable to crush nutshells, pieces of bone, and other jagged bodies. The impaction sometimes causes localized swelling, which should be allowed to subside, before an effort is made at extraction. Brandeis removed a thread from the larynx by means of a brush dipped in mucilage.

Adults will not need anæsthesia, but children, as a rule require it. It is not easy to make a laryngoscopic examination in young children, but the index finger inserted in the larynx will be of valuable assistance.

Tracheotomy may have to be resorted to for the relief of dyspnœa, to reach and remove a foreign body in the larynx, to secure better access to the lower respiratory tract, and sometimes, but rarely, as a precautionary measure. Low tracheotomy is usually the operation to be preferred, and should also be done in addition and previous to direct opening of the larynx; sometimes high tracheotomy, crico-thyrotomy, or direct thyrotomy are necessitated by the situation of the substance in the larynx. Access having been obtained to the foreign body, it may, according to its location, be removed by manipulation through the mouth, or be forced upwards by the insertion of instruments in the tracheal opening. When situated in the trachea or bronchi it is often expelled at once, or forced up so as to be within reach of the forceps. Inversion of the body or succussion may be needed to detach the cause of trouble, and should this fail, probes and forceps may have to be passed directly down into the trachea. The edges of the incision are to be kept widely open, to facilitate manipulation, by

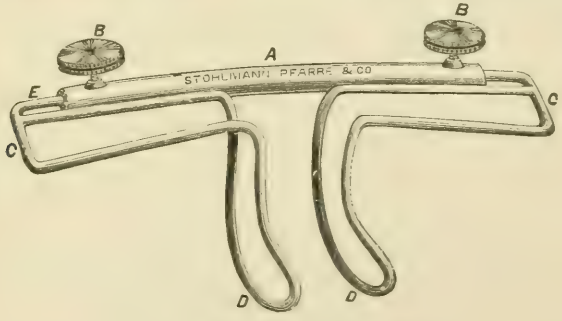


FIG. 83.—Minor's Tracheal Retractor.

means of Labord's dilator or Minor's retractor (see Fig. 83), by threads inserted in the tracheal rings and drawn apart, or, as in a case reported by Wyeth, the edge of the wound may be stitched to the integument.

The finger, a slender probe bent at right angles, or exploratory forceps may be used in exploring the trachea. Fig. 84 shows an

instrument used by Gross. Cohen uses shouldered forceps. Jacobson indorses Störck's laryngeal-tube forceps, which can be bent as desired, being made of German silver. Fig. 85 shows Seiler's tube forceps, which is to be commended. A slender silver probe bent

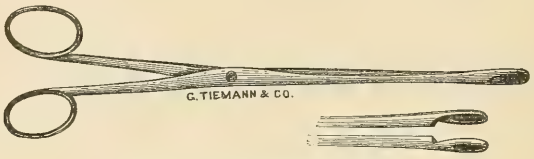


Fig. 84.—Gross' Tracheal Forceps.

into a hook at the end will sometimes suffice to disengage a jagged object from the soft parts, after which, as a rule, it is easily expelled.

A small mirror inserted into the tracheal wound will admit of further inspection by reflected light. The finger may be inserted in the trachea to find or dislodge a foreign body and will reach to the bifurcation.

It is not well to prolong unduly the efforts at exploration and removal after tracheotomy, for the patient is apt to become exhausted, and there is no danger in postponing the procedure for one or several days. Gross advises that not more than three attempts of one minute each should be made. To prevent interference with volun-

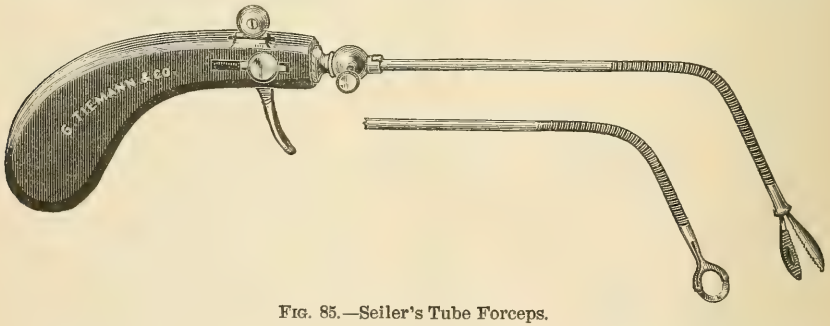


Fig. 85.—Seiler's Tube Forceps.

tary expulsion through the tracheal opening, which may occur, no tube should be inserted, but the edge of the wound may be stitched to the integument.

If tracheotomy and the subsequent manipulations fail to extract a foreign body from the bronchus, an expectant plan of treatment is the wisest for a while, since voluntary expulsion of the substance may occur, and because grave morbid processes may be set up by too much surgical interference. Nesiloff recommends an operation for exposing the bronchi, but the risks are so great that it should be resorted to only as an extreme measure.

FRACTURE OF THE LARYNX.

A fall, a direct blow, or pressure upon the larynx are the usual causes of this very rare accident, which may involve one, two, or more cartilages, and which causes symptoms of serious significance.

Etiology.

Garroting, hanging, choking, a blow of the fist, a bullet or other flying missile, a fall upon some projecting object, even great muscular action in coughing, have been known to cause fracture of the larynx.

In spite of the widely held view that the ossification of cartilages of old age renders one more liable to this injury, the majority of cases reported have been in persons under fifty years of age, and in seven instances it occurred in the first decade.

Symptomatology.

Lateral compression causes an undue projection of the laryngeal prominence, but the usual effect produced by fracture is a depression. There is likely to be much external swelling, resulting directly from the blow, or, where the fracture extends through the lining membrane of the larynx, being caused by emphysematous infiltration. Rupture of blood-vessels allows of an escape of blood into the air passages or the submucous tissues, which causes dyspnœa and the expectoration of frothy, blood-tinged mucus. Respiration may be affected at once or not until several days after the accident. There is usually pain on swallowing, and a cough, but the dyspnœa and bloody sputa are the chief symptoms.

Durham's report shows fracture of the thyroid cartilage alone in 24 cases, the cricoid in 11, thyroid and cricoid in 9, thyroid, cricoid, and trachea in 2, thyroid and hyoid in 4, thyroid, cricoid, and hyoid in 2, cricoid, trachea, and hyoid in 1. In 7 the exact location is not given. The fracture may be simple, linear, comminuted, or compound.

Diagnosis.

The history and evidence of injury, flattening of the prominence, dyspnœa, expectoration of bloody sputa, besides recognition of the deformity, the crepitation of fragments, and the crackling of emphysema upon palpation should serve to clear up the diagnosis. Distortion of the laryngeal cavity and submucous extravasation or escape of blood into the respiratory tract will be shown by the laryngoscope.

Prognosis.

Fracture of the larynx is a serious accident. When the thyroid cartilage alone is injured the danger is lessened; but crushing of the cricoid is usually fatal. Out of 62 cases of laryngeal fracture there were 12 recoveries; in 6 of these the thyroid alone was involved, in 2 the thyroid and hyoid, in 4 the part injured was not mentioned.

Laryngeal obstruction occurs immediately after the injury, and is due either to derangement of the fragments, to submucous extravasation, or to acute inflammation or œdema. A suppurative process is likely to occur at a later stage, and the emphysema may extend even as far down as the mediastinum. Death may result from suffocation, or later from pneumonia, pleurisy, pulmonary œdema, mediastinal abscess, septicæmia, or some other complicating disorder.

Treatment.

Counter-irritants, leeching, or cold dressings should be applied at once to overcome inflammatory action. Displaced parts should be restored by manipulation and kept in place by strips of adhesive plaster, and rest to the organ should be secured by absolute non-use of the voice and avoidance of deglutition. Food may be given through a tube or per rectum. Dyspnœa being the chief source of danger, preparations should be made for tracheotomy upon the first symptom of laryngeal stenosis. The cannula should be inserted as low down as possible.

Restoration of the parts and the preservation of function next demand attention, for the effecting of which a probe may be used, if a tracheal cannula is in position, or bougies may be inserted, or possibly a thyrotomy may have to be performed. Subsequent cicatrization may narrow the larynx and necessitate permanent wearing of the tube, in which case intubation will probably be the best procedure.

PROLAPSE OF THE LARYNGEAL VENTRICLES.

A number of well-authenticated cases place the occurrence of this accident beyond question, although it is one that could scarcely be anticipated. Moxon first discovered it in the post-mortem examination of a man who died from gastric cancer, in whom laryngeal disease was not suspected. Lefferts was the first to meet with it in life. Several other observers have since reported cases, one of which apparently occurred immediately after violent coughing, the majority, however, being of gradual development.

It seems to me that coughing, which is the only direct cause

known for the accident, must produce its effect upon a previously relaxed mucous membrane. It is theoretically possible that the attachments of the thyro-arytenoid muscle are severed by the violence of the act, and with them the attachment of the mucous membrane lining the ventricle. Tuberculosis existed in eight of the cases reported, syphilis in two.

Impairment or loss of voice, and some dyspnœa if there is much swelling, would be the symptoms caused by the accident. The tumor is a smooth, round, or spindle-shaped mass, pale pink or slightly injected, and lies upon the vocal cord, apparently emerging from the ventricular fissure.

Its outline is regular, and it is soft and easily indented, which should serve to distinguish it from the hard, dense, irregularly nodulated, non-pedunculated fibroid tumors, which moreover never spring from the laryngeal ventricle. The long duration of the disease and the absence of ulceration or glandular involvement will exclude malignant origin of the tumor.

We cannot hope to replace successfully a prolapsed ventricle, but the swelling may be somewhat reduced by the local application of astringents. Removal of the prolapsed mass is the only method of entirely restoring the voice, and may be done through the natural passages by means of a snare, or thyrotomy may be performed. Jellenfy reduced the prolapse by superficial incisions over the contour of the mass to shut off the circulation, treating it in effect as a sort of incarcerated hernia.

TUMORS OF THE LARYNX.

Benign Tumors.

The gravity attached to this form of disease is shown by the great amount of space devoted to its consideration in the literature of laryngeal affections. It seems to me that this importance has been somewhat exaggerated, since I cannot regard a benign tumor of the larynx as a grave menace to life, although it does seriously impair phonation, which is, however, not a vital function. Loss of voice, especially when due to a tumor in the larynx, fills the minds of the great majority of people with terror, and yet its main gravity consists in its interference with occupations such as singing or public speaking.

The development, progress, and symptoms of the various benign new growths of the larynx are practically identical. In the order of frequency we meet with papillomata, fibromata, cystomata, myxomata, adenomata, lipomata, angiomata, enchondromata, and mixed tumors.

Etiology.

A hyperæmia of the mucous membrane or a catarrhal inflammation is usually considered to be the cause of a laryngeal neoplasm, but the fact is that it is difficult in any given case to trace its origin. Many of the tumors not only develop in a perfectly healthy larynx, but also cause no abnormal process in the adjacent tissues. Overuse of the voice may be considered a somewhat frequent cause of the disease, occurring among professional singers, actors, etc. Exposure to cold, the inhalation of irritative vapors, eruptive fevers have all been assigned as etiological factors, and one observer reports several cases of warty growths in the larynx, which he considers due to a "papillomatous diathesis," inasmuch as the patients had warty growths in other parts of the body. The vegetations of syphilis and tuberculosis should be regarded as local manifestations of the constitutional disease rather than as distinct tumors.

Mackenzie reports a case of tumor of the larynx in a deaf-mute, which certainly was not occasioned by overuse of the voice.

Some instances are known where the affection occurred before the age of five, one at eighty-two, but the great majority of patients are in the fourth or fifth decade. Men are more liable than women. A number of cases of congenital origin have been observed, especially of the papillomatous variety.

Symptomatology.

Phonation and, more rarely, respiration are interfered with by the presence of benign tumors, which, in the great majority of cases, are situated either upon the vocal cords or near enough to them to prevent their free vibration. In Mackenzie's 100 cases, the voice was affected in 92 instances, entirely lost in 55, and merely hoarse in 37; in 52 a voice change was the only symptom. Fauvel found the voice affected in every one of 300 cases. The conversational voice is not always seriously impaired, but is apt to be weak and easily fatigued, even where the growth is small and situated near, rather than on, the vocal cord. Reflex disturbances are rarely occasioned, so that cough is not often a symptom.

The size of the growth and its situation relative to the chink of the glottis will in a measure determine the amount of interference with respiration. Dyspnœa is usually more marked during inspiration than during expiration, because a growth lies upon the cords in such a way that it falls over the rima glottidis, interfering with the entrance of air, but is lifted up by the outgoing air. A sessile, immovable growth will of course affect both respiratory acts.

The growth sometimes occasions spasmodic contraction of the laryngeal muscles, especially in young children. A favorite situation for pedunculated growths is near the anterior commissure of the vocal cords; by falling below the glottis they often occasion a dyspnœa which ceases when they are blown out, as it were, upon the superior surface of the cords. In young children, the growth will sometimes produce a catarrhal laryngitis, causing attacks of dyspnœa at night.

Severe hemorrhage would indicate a malignant growth rather than a benign tumor, but the sputa may become tinged with blood from erosion of a papilloma situated near the vocal cords and subject to friction from motion of the parts. Where the growth is large, there may be a sense of fulness, causing discomfort but rarely pain. Perversion of the sense of taste with hypersecretion of saliva has been reported.

Pathology.

Papilloma.—This is the most frequently occurring form of laryngeal tumor. Out of 1,100 cases collated by Bruns 54 per cent. were of this variety, as were 67 per cent. of Mackenzie's and 69 per cent. of Fauvel's. One hundred and sixty-three of 310 cases noted by Elsberg were papillomata. Virchow calls this variety of growth "pachydermia verrucosa," in contradistinction to "pachydermia diffusa."

Both forms of the disease have their origin in the papillæ of the mucosa; in the diffuse form, hypertrophic changes occur in the deeper tissues. In pachydermia verrucosa, or papilloma, not only the papillæ are affected but the epithelial structures on the surface of the mucosa as well. There is a localized proliferation of epithelial cells, which instead of losing their vitality become heaped up into a wart-like growth.

Small rounded projections are seen upon the surface of a papilloma, each one probably marking the site of a papilla of the normal mucous membrane. If cut longitudinally, one of these projections will show at its centre the long papilla containing the original vascular loop, supported by loose connective tissue, the whole surrounded by fifteen to twenty layers of epithelial cells.

The favorite situation for these growths is the anterior portion of the vocal cords, although in some few cases they are found upon the ventricular bands, the aryepiglottic folds, and the epiglottis. One would be inclined to think that the arytenoid commissure, which is subject to constant motion, might naturally be the seat of warty growths, but it never is, and the presence of a tumor in that position or on the posterior part of the cord would, on the contrary, suggest a malignant growth.

Papillomata may be pedunculated, but are usually sessile, occur singly or in groups, and in size vary from a millet seed to a tumor filling the supraglottic laryngeal cavity.

The tumors develop with great rapidity in children, but rather slowly in adults. The involvement of adjacent papillæ causes a gradual broadening of their base. All forms of benign tumors in adults tend to confine themselves to the portion of the larynx above the glottis, but in children they sometimes extend below.

Fibroma.—Next in frequency to the papillomata, these growths occur in eight to ten per cent. of all tumors observed, and belong especially to adult life. In common with fibrous growths elsewhere, these are composed of a network of fibres, enclosing a number of branching cells, are poorly supplied with blood-vessels, but invested by a mucous membrane which shows signs of hyperæmia. A well-marked areola surrounds the growth. Fibromata rarely have a pedicle, and many cases so recorded are probably of a cystic character. One or other of the vocal cords is the favorite site for a fibroma, which occasionally causes the development of a second growth upon the other side. They are not multiple. They are small, rounded, smooth (except where multilobular), and vary in size from a millet seed to a hazelnut. In one case the growth originated upon the upper border of the cricoid cartilage and almost entirely filled the supraglottic larynx.

Cystoma.—This form of growth is of frequent occurrence, although, probably from mistaken diagnosis or non-observation, it used to be considered extremely rare. It occurs between the ages of twenty-five and fifty, as a rule, but has been reported at fourteen and at sixty-four.

Obstruction of the duct of a mucous gland, from which follow dilatation of its cavity by the retained secretion and its projection above the surface of the mucous membrane, is the usual cause of the tumor, but it is not yet known whether this obstruction is due to degeneration of the epithelial lining of the gland, atrophy of the duct, or degenerative changes in the cells themselves without affection of the duct.

Moure found that out of 117 cases the tumor sprang from the epiglottis in 50, the vocal cords in 45, the ventricles of Morgagni in 8, the arytenoid cartilages in 4, the aryepiglottic folds in 3, the cartilages of Santorini in 1, and the ventricular bands in 2. In 4 the origin was unknown.

The tumor is round, smooth, easily compressible, and movable, varies in size from a pinhead to a hazelnut, and is covered with mucous membrane of a light-red color.

Myxoma.—These growths, which are of somewhat rare occurrence, are invariably found upon the vocal cords; they may be pedunculated and multilobular, or sessile. They are the result of a species of myxomatous degeneration of the mucous membrane. Sometimes they are smooth and of a gelatinous aspect, like the myxomatous tumors met with in other parts of the respiratory passages, and again they are mammillated, grayish pink in color, and so like the papillomata that the microscope alone can distinguish their true nature. They are usually unilateral, but in some cases both cords are affected. They are composed of a loose meshwork of connective-tissue fibres holding branching myxomatous cells.

Angioma.—As a rule, these vascular tumors spring from the vocal cords, but they have been known to originate from the ventricular bands, the epiglottis, the hyoid fossa, and the lingual sinus. Their size is from that of a pea to that of a hazelnut; their structure is a mass of blood-vessels held together by loose bands of connective tissue. They occur in adult life and are almost invariably unilateral.

Chondroma.—The favorite seat of this form of growth is the cricoid cartilage; the next the thyroid, the epiglottis, and the arytenoid. These tumors always extend inwards, forming a large, sessile, immovable mass which gives rise to dyspnœa. They are of slow growth and belong to adult life. Their outline is irregular, and they are covered with a mucous membrane which is somewhat hyperæmic and liable to become eroded by friction; they are composed of hyaline cartilage, except when situated upon the epiglottis, where fibrous tissue enters into their structure as well. They may be of the size of a cherry pit, or may completely fill the laryngeal cavity.

Adenoma.—It is not probable that this variety of new growth is ever found in the larynx, although a few such cases have been reported; in these, however, the diagnosis was not beyond question.

Lipoma.—With the exception of one case reported by Bruns, fatty tumors in the larynx originate only in the aryepiglottic fold and fall externally into the hyoid fossa. The exceptional case was intralaryngeal. Histologically these growths resemble similar neoplasms found elsewhere.

In our description of laryngeal tumors we have classified them under the head of the histological element which forms the chief part of their composition, but other tissues are often mingled with the prevailing type.

Diagnosis.

A peculiar character of the voice will sometimes indicate a laryngeal tumor to a trained ear, but the laryngoscope alone will make the

diagnosis certain, and show the size, location, and often the character of a growth.

A *papilloma* (see Figs. 86, 87, 88) is soft, moves slightly during breathing and speaking, is grayish-white or pinkish-white in color, has a finely mammillated or wart-like contour, and as a rule originates from the anterior portion or angle of the vocal cords. Epithelioma in its early stages is the only tumor with which it is likely to be confounded, but this occurs at a later age than papilloma, which belongs to early life.

A *fibroma* is usually non-pedunculated, embedded in the neighboring tissues, and almost always originates from the anterior surface of the vocal cords. It is a hard, resisting mass, round or irregularly nodulated, and covered with a more or less highly injected mucous membrane. It does not resemble a myxoma, but is somewhat like a

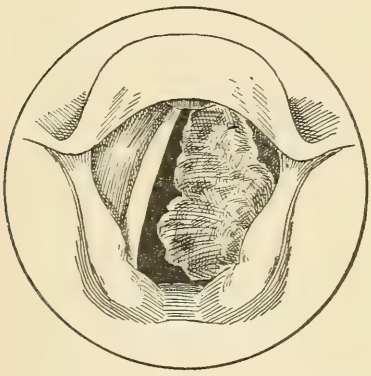


FIG. 86.—Papilloma of the Right Ventricular Band.

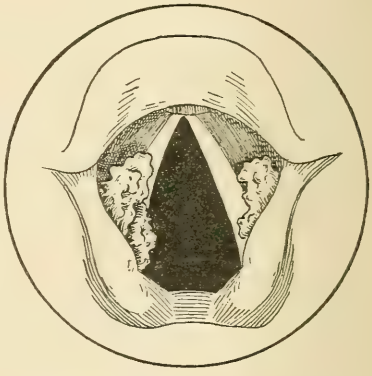


FIG. 87.—Papilloma of the Ventricular Bands.

chondroma, which, however, is never found upon the vocal cords, or a cystoma, which differs in being semi-translucent, soft, compressible, usually movable and pedunculated. The progress and clinical history of a gummy tumor should prevent its being mistaken for fibroma.

A *cystoma* on the cords forms an almost translucent sac, but when it originates from the parts above (see Fig. 89) is somewhat vascular, which renders it reddish and more opaque. It is small, soft, and compressible, and collapses when seized with the forceps.

A *chondroma* (see Fig. 90) is especially distinguished by its slowness of growth. It is hard, dense, and resistant, covered by normal mucous membrane, and springs from any of the laryngeal cartilages, preferably the cricoid. Here it may be mistaken for carcinoma, which, however, rarely appears in early life, and is rarely seen

below the glottis; or for a perichondritis, which differs from it in the suddenness of the onset, the acute symptoms of local inflammation, and the early development of dyspnœa.

Chondroma from a cartilage above the glottis somewhat resembles

FIG. 88.—Papilloma of the Ventricular Band Completely Filling the Vestibule of the Larynx.

FIG. 89.—Cystoma of the Epiglottis.

fibroma, but the clinical history should point to its origin and character. The softer tumors, such as myxoma and cystoma, would of course not be confounded with a growth of such density as a fibroma.

Myxoma.—This growth in the larynx somewhat resembles myxoma of the nasal cavities, especially where it is broadly sessile, but when multiple it is more like papilloma. The microscope must es-

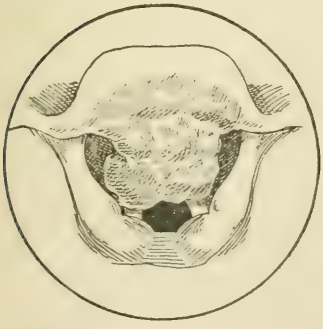


FIG. 90.—Chondroma of the Epiglottis.

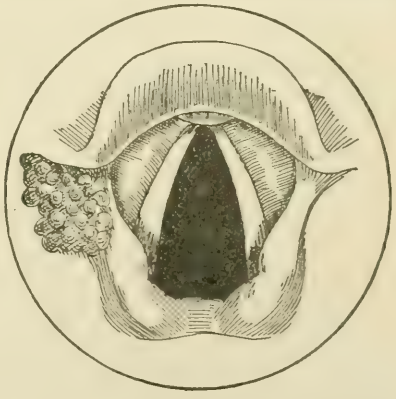


FIG. 91.—Angioma of the Left Aryepiglottic Fold.

tablish the diagnosis. As a rule it is soft and of a grayish-white color, and almost always originates on the vocal cords.

Angioma (see Fig. 91).—It would be difficult to make a mistake

in the diagnosis of this raspberry-like mass of highly injected blood-vessels.

Prognosis.

Unless these tumors become so large as to interfere with respiration, they do not threaten life, and even then tracheotomy can prevent a fatal result, the dyspnœa being always so slowly progressive as to give ample warning.

The most rapidly developing laryngeal tumor is the papilloma, especially in very young children, and yet even with these there is little danger of suffocation.

Other portions of the larynx are but seldom subjected to disturbance by the presence of a new growth; an occasional laryngitis, spasm of the glottis, or œdema may occur but is not productive of danger.

In this era of surgery the prognosis is good. Papilloma alone of the benign tumors exhibits a tendency to recurrence, and this probably because it is so difficult to remove every portion of the neoplasm by the endolaryngeal operation chiefly used in this affection. The voice may become permanently affected from injury to the tissues in the removal of a large tumor, but as a rule the prognosis regarding restoration of the voice is good. A few cases have been reported of spontaneous expulsion of the growth.

The question of malignant degeneration of a benign neoplasm has been considerably agitated, but Semon has definitely answered it by his investigation of reported and personal cases. Of 2,531 instances of benign laryngeal tumors which were not operated upon, malignant degeneration occurred in 12, or 1 in every 211 cases; and in 8,216 cases subjected to operation, degeneration afterward took place in 33, or 1 in 249 cases. The tendency to degeneration, which is chiefly confined to papillomatous growths, is not only extremely feeble, but is diminished by operative interference.

Treatment.

In the earlier days, before the invention of the laryngoscope, a neoplasm, if recognized at all, was removed by an external opening through the neck. Now that even small growths are visible to the eye, they are removed largely through the natural passages by means of evulsion, crushing, abscission by means of knives or scissors, écrasement, or the use of the chemical or potential cautery.

In evulsion, the growth is seized by the bladed forceps or tube forceps, and torn from its site. Fig. 92 shows the Mackenzie blade forceps, which is meant to work antero-posteriorly or laterally and

in which the blades are bent at a right angle, differing in this from Fauvel's forceps, in which the blades are curved to the segment of a circle and are fenestrated to obscure a view of the parts as little as possible. The Mackenzie instrument is easier to use, but I agree with Fauvel that the lateral movement is of advantage. Many tube forceps have been invented, but I consider them too delicate in construction, and the movements of the blades too limited for any but

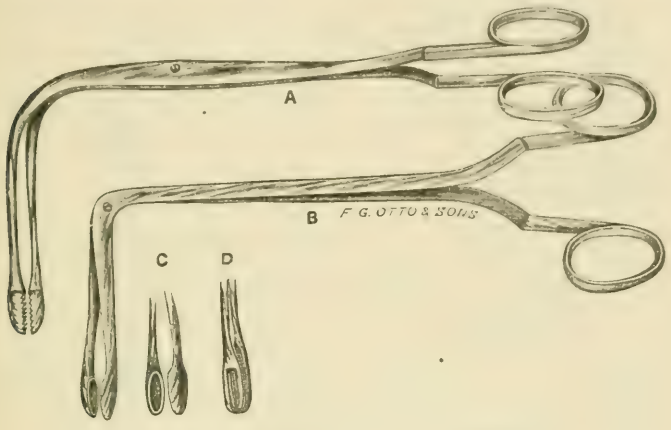


FIG. 92.—Mackenzie's Laryngeal Forceps.

the smaller growths. Störck's instrument may be seen in Fig. 93 and it will be noted that the distal extremity of the tube is bent to the quadrant of a circle. In Schrötter's instrument (Fig. 94) the handle is thrown to one side, out of the line of vision, by the horizontal and vertical curving of the tube: it is a convenient instrument, but of somewhat limited use. I think the Mackenzie tube forceps, shown in Fig. 95, too slender, and Gottstein's instrument (Fig. 96) is not easy to handle.

The same instruments that are used for evulsion will answer to crush the growths. By abscission we mean cutting through the base of the tumor with a concealed knife or small scissors, such as may be seen in the illustrations of the Störck and Schrötter instruments.

Only such growths as project into the larynx in such a way that a wire loop can be placed about their base will admit of écrasement. Schrötter's instrument, or an ordinary snare, bent to a suitable curve, may be used.

Excision by means of the guillotine, shown in the illustration of Störck's instrument, is applicable to nearly the same class of cases as the snare.

Voltolini advises the forcible introduction of a sponge probang

into the larynx to dislodge growths, and this method is adapted to small pedunculated tumors attached near the edge of the vocal cords.

The cautery may be used to destroy laryngeal growths. Chromic acid seems to me preferable to other caustics; it may be fused on the end of a curved probe, or a hooded porte-caustique may be employed.

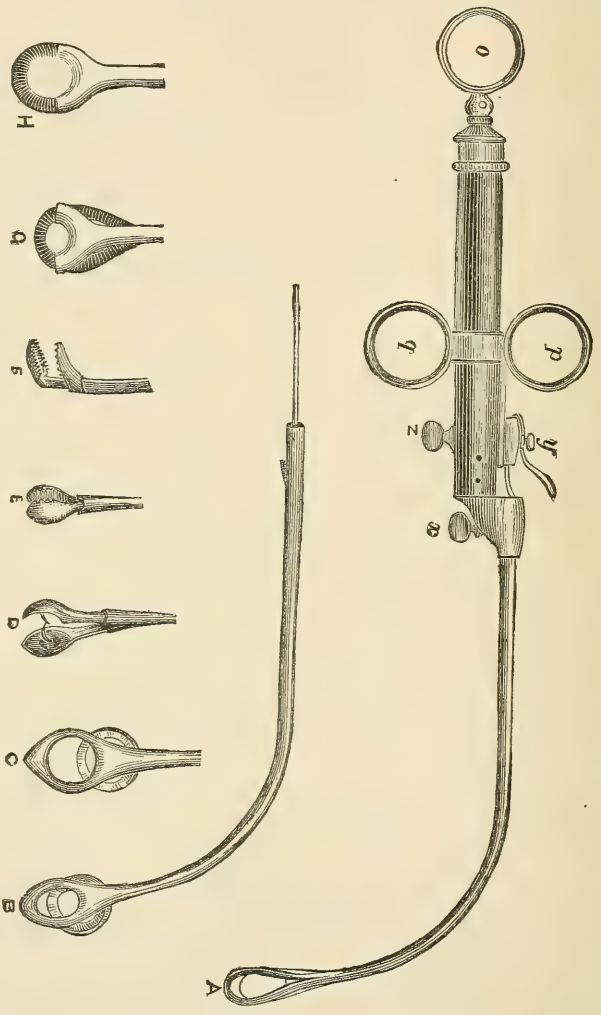


FIG. 93.—Störck's Forceps. A, wire écraseur; B and C guillotines; D, E, and F, forceps; G, guillotine half closed; H, the same open.

A simple procedure, which I have often made use of, is the wrapping of a piece of cotton on the end of a laryngeal probang, a saturated solution of chromic acid being placed on that part of the cotton which comes in contact with the growth. I consider nitrate of silver to be inefficient for the purpose, but powerful caustics, such as caustic

potash, Vienna paste, or nitric acid, should not be used in the larynx.

Where the milder chemical cautery is of no use, the galvano-cautery is the best of all methods, in that one can place the electrode upon the desired spot before developing the heat, and shut off the

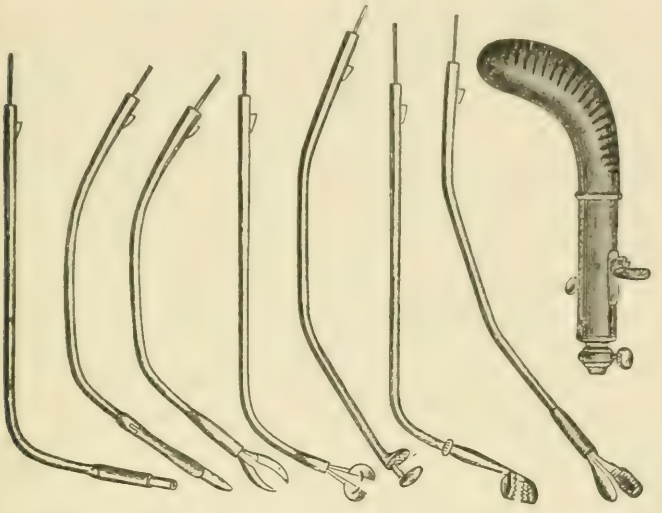


FIG. 94.—Schrötter's Forceps with Concealed Knives and Ap

heat before removing the electrode. In the majority of cases, it will be best to use the smaller-sized platinum tips, and nicely adjust the strength of the current.

Training of the parts by means of probes has been rendered unnecessary by the introduction of cocaine for local anæsthesia. A twenty-

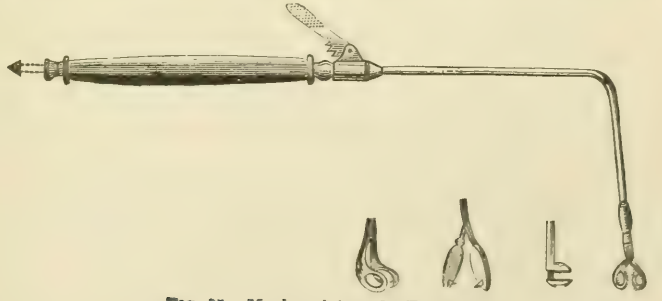


FIG. 95.—Mackenzie's Tube Forceps.

per-cent. solution should be freely sprayed on the fauces and larynx, and its action tested three or four minutes later by passing in a pledget of cotton saturated with the solution. When complete anæsthesia is established, which should be in about five minutes, the operation

should be proceeded with without delay, as the action of the anæsthetic in the larynx can be depended upon for only eight or ten minutes. Involuntary muscular contractions of the fauces which might interfere with the operation may be partially overcome by having the patient swallow small pieces of ice in the intervals between the applications of cocaine.

The patient now protrudes his tongue and holds it with a napkin between the thumb and forefinger of his right hand. The laryngeal mirror is introduced, held in the left hand of the operator, the patient's head being thrown well back, and supported upon a head-rest. As soon as the tumor is visible, the forceps, which has been warmed, is taken in the right hand, and the beak is passed into the fauces nearly to the pharyngeal wall and then carried down into the larynx, care being taken that the walls of pharynx or larynx are not touched. The mirror, if held firmly in position, will clearly show the neoplasm and the beak of the instrument.

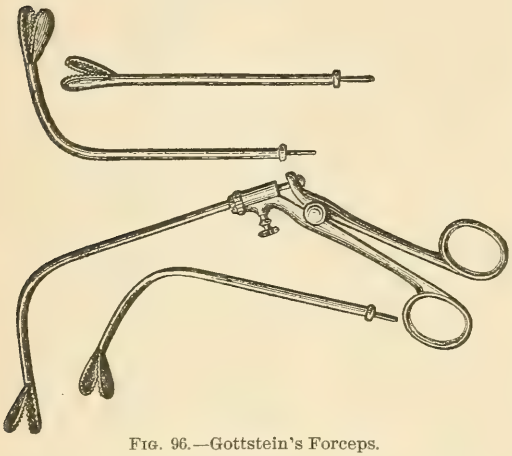


Fig. 96.—Gottstein's Forceps.

If the tumor is situated above the glottis, just before the beak of the instrument is turned into the larynx, the patient should be told to take a full-breath, and then utter a high-pitched, prolonged "*a*," which will not only divert his attention, but raise the epiglottis as far as possible and fully expose the laryngeal cavity. The phonatory position of the larynx is usually the best for the seizure of the growth, although in some cases this may be done to better advantage during inspiration or expiration.

The thyro-epiglottic muscle is very apt to contract firmly and shut off all view from the larynx upon the insertion of the instrument, wherefore great care must be exercised to avoid touching the laryngeal walls.

The depth of the larynx varies greatly in different individuals, a fact to be borne in mind in selecting blades of sufficient length to reach the growth without touching the epiglottis with the shaft. Of course the forceps is to be closed when introduced, and only opened at the moment of seizure of the growth.

No operation in the throat requires greater skill, dexterity, and experience; this is best gained by practice upon a cadaver. The Schrötter tube forceps is probably the best for removal of a small papilloma, but the stout Mackenzie or Fauvel bladed forceps is to be preferred for larger growths. The latter are usually removed piecemeal at intervals of from three or four days to a week.

A snare is the most useful means of taking away a fibroma which projects into the laryngeal cavity, but the cutting forceps or curette will be needed to remove a deeply embedded tumor. If small and inaccessible it is best not to interfere with them. Evacuation of the sac of a cystoma by means of the concealed or naked knife will cause its disappearance. Myxomata should be seized and torn away by the bladed forceps. Where possible, it is better to remove an angioma by the use of the wire loop, for evulsion may set up a troublesome or dangerous hemorrhage. Even the snare is not entirely devoid of this danger, so that possibly the galvano-cautery would be the safest method for its destruction. If the tumor is of large size, thyrotomy, with or without a preliminary tracheotomy, would be best. Cauterization will suffice for the treatment of small enchondromata, but larger growths should be removed by thyrotomy.

The *after-treatment* of removal of a laryngeal tumor consists in the application of caustics at intervals of a week or longer, to destroy any small fragments which may remain, or to prevent recurrence, especially in the case of papillomata. Chromic acid fused on a probe is undoubtedly the best agent.

The galvano-cautery is indicated where more active measures are demanded. An external operation will be needed for cases in which the growth cannot be gotten rid of by endolaryngeal means. Thyrotomy, subhyoid pharyngotomy, and infrathyroid laryngotomy are the operations used. By the first the larynx is most easily reached, but there is some danger to the patient, and the voice is liable to be seriously impaired. For this reason the two other operations are probably to be preferred, but, unhappily, they are rarely available.

I will here, as in my work on "Diseases of the Nose and Throat," quote certain propositions laid down by Browne:

First. Attempts at removal of growths from within the larynx are not in themselves so innocuous as is generally believed; but, on the contrary, direct injury to the healthy parts of the larynx, leading to even fatal results, is by no means of infrequent occurrence.

Second. The functional symptoms occasioned by benign growths in the larynx, are in a large proportion of cases not sufficiently grave to warrant instrumental interference.

Third. Many of those new formations will disappear, or be re-

duced by appropriate local and constitutional medical treatment, especially when of recent occurrence.

Fourth. Recurrence of laryngeal growths after removal *per vias naturales* is much more frequent than is generally supposed.

Fifth. While primary malignant or cancerous growths are of rare occurrence within the larynx, benign growths occasionally assume a malignant and even cancerous character, by the irritation produced by attempts at removal.

Sixth. The instruments most generally now in use are far more dangerous than those formerly employed.

Seventh. The cardinal law that an extralaryngeal method ought never to be adopted unless there be danger to life from suffocation or dysphagia, should be applied with equal force to intralaryngeal operations, and it is a subject worthy of consideration whether in many cases tracheotomy alone might not be more frequently performed, first, with a view of placing the patient in safety when dangerous symptoms are present; second, in order that the larynx may have complete functional rest; and third, as a preliminary to further treatment, radical or palliative.

These statements are perhaps too radical to be fully indorsed, but they carry so much of suggestion and come from so excellent an authority that they cannot be omitted.

Sarcoma.

This affection is of comparatively rare occurrence, and, as compared to carcinoma, is met with in the proportion of 1 to 612.

Etiology.

Our conclusions are based upon the study of 47 cases, 46 reported by Mackenzie and 1 occurring in my own practice. Thirty-four were in men, 13 in women; the earliest age was nineteen, the oldest seventy-four, but the majority of cases occurred in the fifth and sixth decade.

The disease does not seem in any instance to have been traceable to heredity. Local inflammatory troubles preceded it in many cases, yet we cannot really consider catarrhal laryngitis to have been in any way a cause. The disease as a general thing develops in patients apparently in good condition and without known cause.

Pathology.

Histologically the growth is like that of sarcoma elsewhere. Three of the cases were alveolar in character, 5 round-celled, 6 spindle-celled, 2 mixed-celled, 2 lymphosarcoma, 4 reported as papillary

sarcomata, with 1 each of fibrosarcoma, adenosarcoma, and fibromyxosarcoma.

The vocal cords were the seat of disease in 16 cases, the ventricular bands in 9, the right ventricle in 2, the epiglottis in 5, the right pyriform sinus in 1. In my case and one other the origin was subglottic.

The growth, as a rule, confines itself to the laryngeal cavity, but occasionally it seems to possess a downward tendency, the trachea having been known to become involved. In one case the disease started in the tonsil and pharynx, in one in the commissure. A unique case is that in which the larynx, pharynx, soft palate, and base of the tongue were invaded. As a rule the disease is unilateral, but it has been known to develop in both sides of the larynx.

Symptomatology.

The first symptom is usually hoarseness or complete aphonia, followed by respiratory troubles as the air passages are encroached upon. Cough, excited by a secretion from the surface of the growth, is a symptom, but is rarely very troublesome unless the growth extends below the glottis. If the epiglottis is involved there will be difficulty in swallowing. Erosion and ulceration from attrition occur early, but do not cause severe hemorrhage, although the sputa may be tinged with blood. Pain is not often occasioned.

Rapidity of growth varies; in one case the tumor filled the larynx at the end of two months, in another at the end of four months.

The cervical glands are rarely the seat of infiltration, the tendency to spreading being very feeble. Only one case is known where the generalization extended farther than the cervical glands; in this instance the lungs, liver, and brain were involved, causing death three months after the primary invasion of the larynx. A form of cachexia occurs after the growth has been present for a considerable time, but is less pronounced than in carcinoma.

Diagnosis.

The term grumous best describes these soft, irregularly rounded, pinkish or more often grayish, semi-opaque tumors. They cannot be absolutely distinguished from carcinomata by the eye, but to the practised eye their malignancy is evident, as shown by the unhealthy aspect, thick, adherent ropy muco-pus, irregular outlines, and distortion of parts. Points of erosion are seen, but ulceration seldom occurs.

Absence of involvement of the cervical glands would point to sar-

coma rather than carcinoma, and yet it is not to be forgotten that they are involved only in the later stage of cancer. The microscope is the only sure means of diagnosis.

Prognosis.

The disease almost invariably leads to a fatal issue. Of the 47 cases reported the histories are incomplete, but 18 deaths are known to have occurred, the average duration of life from the beginning of the disease having been a little over nineteen months. Recurrence caused death in 6 cases, suffocation in 4, pneumonia after removal in 2, hemorrhage after extirpation in 1, generalization in 1, congestion of the lungs and nephritis in 1, asthenia in 3.

Treatment.

The results of operative measures indicate that extirpation is the treatment most likely to insure success. The choice of an operation must depend upon the conditions in the given case. In the early stages, while the tumor is small, it will be best to attempt to remove it through the natural passages; if this fail, thyrotomy should be resorted to. Resection or complete extirpation of the larynx may become imperative to save the life of the patient.

Carcinoma.

Out of 11,131 cases of cancer recorded in the hospitals of Vienna only 63 affected the larynx. Lébert found 3 instances of laryngeal cancer in 9,118 cases, Winniwarter 1 out of 548, and Baker 3 in 500. These statistics show how rarely the larynx is the seat of carcinomatous disease.

Etiology.

Heredity is found to have had a marked influence in from twenty to twenty-five per cent. of the cases reported. Men are more subject than women to the disease, which belongs to the later years of life, occurring most frequently in the sixth decade. Overstrain of the voice and laryngeal catarrh have sometimes been supposed to be the starting-point of the disease, but they were more probably caused by it.

Pathology.

Epithelioma is the most frequent form of cancer found in this situation. Any part of the larynx may give rise to the tumor, but the site of predilection appears to be the ventricular bands. Intrinsic growths, those confined to the larynx proper, show little tendency

to spread to the lymphatic glands of the neck. Extrinsic cancer, that affecting the epiglottis, aryepiglottic folds, or the arytenoids, may extend to the lymphatics at an early period.

Anatomists have demonstrated so rich a capillary network of lymphatics in the submucous tissues in both the extrinsic and intrinsic portions of the larynx, connected so intimately with the lymphatic glands of the neck, that it is difficult to understand why intrinsic cancer has so little tendency to involve these glands. But Sappey has shown that as the lymphatics reach the larynx they spread out into a close network, which becomes more and more attenuated until, in passing from the upper portion of the lining membrane of the larynx, it becomes suddenly impoverished. In this peculiarity may be found the explanation.

Symptomatology.

In the early stages the symptoms are the same as those of a benign tumor, vocal impairment being the first. It may, however, not appear for some time, especially when the ventricular bands are the seat of the disease, but as the growth becomes larger the voice gradually becomes weaker until it is at length almost completely lost.

Dyspnœa is the next symptom, varying according to the degree of interference with the respiratory passages. If the cancer is extrinsic it may in the early stages encroach upon the food tract and cause dysphagia, and also give rise to profuse salivation.

Where the glands are involved, they become enlarged early in the course of the disease. In from three to six months the cancer usually becomes of a considerable size, causing pronounced subjective symptoms, and undergoes ulceration with hemorrhage, the ulceration being partly due to attrition, but chiefly to the increased rapidity of cell proliferation. As a result of interstitial necrosis, there is shown a tendency to the formation of fissured or crater-like excavations, especially in encephaloid cancer, the epithelial variety being distinguished by a more superficial form of ulceration.

The secretion from the part consists of a healthy mucus in rather more than normal amount, which may cause a slight cough, but after ulceration occurs it becomes superabundant, consisting of a thin seromucus containing pus cells and necrotic tissue. Its aspect is unhealthy, its color a grayish-yellow. Coincidently the patient's breath becomes offensive from the peculiar musty fetor which characterizes carcinoma. The sputa may be slightly tinged with blood from the ulceration, or from erosion of an arterial branch there may be serious and repeated bleeding. Only one case of fatal hemorrhage has been reported.

Pain in rare cases is absent, but is usually a prominent symptom, especially in extrinsic cancer. It may be constant or occur during swallowing. In character it is sharp and lancinating, and as a rule radiates toward the ear or over the whole side of the neck.

The lymphatic isolation of the larynx explains the delay and occasional absence of cachexia in cases of cancer of the larynx.

Diagnosis.

No definite diagnosis can be made in the early stage of the disease. Cell infiltration being its chief characteristic, and burrowing deeply in surrounding parts, the disease extends much farther than is shown by even a distinct tumefaction; the impairment of motility of muscles and articulations in conjunction with a tumor should therefore lead to suspicions of malignancy. Later in the disease the whole appearance of the larynx, filled as it is with an irregular, broadly infiltrating mass which causes a distortion of the parts and is accompanied by ulceration, a secretion of a peculiar nature, and a fetid odor, points only too clearly to the nature of the affection. The age of the patient, sometimes the involvement of the cervical glands, the progressive course of the symptoms, the character of the pain, and the presence of cachexia toward the last, also indicate carcinoma.

Prognosis.

Always fatal and most distressing, cancer is even more unendurable in the larynx from its interference with respiration. Baker's statistics tend to prove that the duration of cancer in various parts of the body is from twelve to forty-three months, but Fauvel reports seven cases of encephaloid cancer of the larynx which were not operated upon in which, on an average, death occurred in three years, and six cases where it occurred in one year and eleven months. One would naturally suppose that cancer in the larynx would be more rapidly fatal than in other parts of the body because of the danger of suffocation. That it is not must be due to a slower development in that situation or to early ulceration maintaining a patency of the air passages.

Treatment.

Relief of symptoms, not a cure of the disease, is given by the local application of anodynes and disinfectants as sprays or powders.

For disinfection we may use a twenty-per-cent. solution of peroxide of hydrogen, a one-half-per-cent. solution of pyoktanin, a two-per-cent. solution of permanganate of potassium, or any carbolized alka-

line solution. Iodoform is probably the best substance to overcome the fetor of the ulceration, or iodol may be used.

To relieve pain locally morphine in powder or solution may be used, but no more than the physiological dose should be applied at one sitting. Cocaine is more effective, but less permanent in its effects. A five to ten per cent. solution in an atomizer may be given to the patient for use whenever necessary.

A case is reported in which the daily internal administration of gtt. xv. of tincture of thuja occidentalis, with local applications of the same drug, apparently caused a case of cancer of the larynx to remain stationary for twenty-six months.

Operative interference is usually urgently indicated. If dyspnœa occur, tracheotomy should be promptly resorted to as a palliative measure. From Fauvel's statistics it would seem that this operation prolonged life for nine months in several cases of encephaloid cancer, and for more than two years in a case of epithelioma.

Caustics, endolaryngeal operations, thyrotomy, resection, and extirpation of the larynx are the radical measures in use. Chemical or actual cauterization only serves to stimulate the growth of a cancer or increase the severity of the symptoms, and is therefore to be absolutely condemned.

In spite of its apparent simplicity, the endolaryngeal operation for removal of the growth has been successful in only one case. Out of 22 cases of thyrotomy compiled by Mackenzie, only 2 were successful, both of which were performed by Billroth. One of the cases, a man of forty-five, was well three years later; the other, a man of forty-one, was well two years and nine months later. Butlin reports 2 cases of cure by thyrotomy.

Of resection or partial extirpation of the larynx Mackenzie gathered 35 cases, of which 6 were well at the end of from twelve to eighteen months, and 1 three years after the operation.

Mackenzie found that total extirpation of the larynx had been performed in 138 cases. Of these, 100 died or had a recurrence. Of the remainder the history of some is imperfect, but we may conclude that about 13, or a little less than 10 per cent., were successful.

Thyrotomy can give hope of success only when performed early in the disease. Resection is indicated only in those cases in which the growth is confined to one side of the larynx, and total extirpation is called for when the whole cavity is invaded.

No question is more difficult to decide in the case of laryngeal cancer than that of operative interference, and that of the choice of suitable operation.

If a new growth, in a patient past middle life, shows a disposition

to recurrence, and particularly if the microscope shows a tendency to epithelial formation, thyrotomy should be performed and the growth thoroughly removed. If recurrence take place after this, resection of the larynx should be done.

Arrest of the disease is the most important result to be obtained, relief of the patient the second; resection may not prevent recurrence, but it will probably add many months to the patient's life. Whether total extirpation shall be practised upon recurrence after resection must depend upon the special indications. The surgeon will do well to bear in mind that the fatality of this operation is over ninety per cent.

THE DISEASES

OF THE

TRACHEA AND BRONCHIAL TUBES.

BY

SIR THOMAS GRAINGER STEWART

AND

GEORGE ALEXANDER GIBSON.

THE DISEASES OF THE TRACHEA AND BRONCHIAL TUBES.

INTRODUCTORY.

The diseases of the different portions of the respiratory tract may, like so many others, be said to have been differentiated from each other only during modern times. It is perfectly true that from the days of Morgagni various morbid appearances found in different parts of the tract have been recognized, but the special clinical features presented by the various diseases could not be appreciated until the present century. Our knowledge of diseases of the trachea and bronchial tubes was first definitely established by the work of Badham [1] and Frank.[2] Laennec [3] also soon afterwards made an attempt to systematize the diseases of the respiratory organs, and in his classical work he tries to distinguish different lesions of the trachea and bronchial tubes. The gaps left in our knowledge with regard to these diseases were gradually filled up by the publication of observations by the elder Williams,[4] Stokes,[5] Skoda,[6] and Walshe,[7] and in more recent times by the contributions of Biermer,[8] Lebert,[9] Riegel,[10] and Hamilton.[11] It is not our intention in this article to dwell at any length upon the history of our knowledge of the diseases with which we have to deal. In many of the works to which we have already referred, particularly in those of Biermer [8] and Riegel,[10] there are full bibliographies, and for information in regard to the history of our knowledge of the diseases of the respiratory tubes reference may be made to these works. In the following pages, however, we shall in the different sections make reference, when it seems advisable, to those authors who have in the past more especially elucidated the questions under discussion.

In the present article the description of the diseases of the trachea and bronchial tubes is preceded by introductory discussions as to the anatomical, physiological, pathological, semeiological, and therapeutical aspects of these diseases in general. The diseases themselves are classified in accordance with the usually accepted teaching. The diseases of the trachea and bronchi are dealt with

first, and then those of the bronchial tubes. Among diseases of the trachea and bronchi, the inflammations are first considered—catarrhal, fibrinous, diphtheritic, and mycotic; ulceration and stenosis follow. Of the diseases of the bronchial tubes, as in the case of the trachea and bronchi, the inflammatory affections receive attention in the first place: Catarrhal inflammation in the acute form as it affects the smaller or the larger tubes, and in the chronic form according as it is dry, moist, or putrid; fibrinous inflammation in its acute and chronic forms then follows. Stenosis, or narrowing of the bronchial tubes, and bronchiectasis or dilatation are then taken up, and asthma finally receives attention.

Anatomy.

The larynx lies in front of the third, fourth, fifth, and sixth cervical vertebræ, from the bodies of which it is separated by the pharynx and prevertebral muscles. The trachea begins, as does also the œsophagus, at the sixth cervical vertebra, and extends to the intervertebral disc between the fourth and fifth dorsal vertebræ where it ends by dividing into the two bronchi. The trachea occupies the mesial plane except toward its lower end where it inclines slightly to the right side. The two bronchi differ somewhat in dimensions and course, the right being shorter, wider, and lying more in the axis of the trachea than the left. Passing into the root of the lung the bronchi divide into bronchia, which in turn undergo subdivision until they finally enter the pulmonary lobules as bronchioles. Each bronchiole dilates to some extent to form the infundibulum, which expands into the alveoli.

In front of the cervical trachea in its upper part lie the isthmus of the thyroid gland and the inferior thyroid veins. The sterno-hyoid and sterno-thyroid muscles are more superficial, and in the subcutaneous fascia are the anterior jugular veins with their connecting twigs. Posteriorly the trachea lies against the œsophagus. On each side are the common carotid artery and the lobe of the thyroid gland; and the recurrent laryngeal nerve lies in the angular space between the trachea and œsophagus.

The thoracic trachea is in relation anteriorly to the deep cardiac plexus, the aortic arch with the origins of the innominate and left carotid arteries, the left innominate vein, the remains of the thymus gland, the attachments of the sterno-hyoid and sterno-thyroid muscles, and the manubrium sterni. Posteriorly it rests against the œsophagus, which, however, lies rather to the left of the mesial plane. To the right are the pleura, right vagus nerve, and innomi-

nate artery, while to the left are the left recurrent laryngeal nerve, the aortic arch, and the left subclavian artery.

Each bronchus as it passes into the lung is in relationship to two pulmonary veins, a pulmonary artery, small bronchial arteries and veins, the pulmonary lymphatics, and the pulmonary nerves derived from the anterior and posterior pulmonary plexuses.

The root of each lung, containing these structures, has in front the phrenic nerve with its nutrient artery, and the anterior pulmonary plexus; while on the right side the posterior plexus lies behind, the vena azygos major is above, and the superior vena cava is in front; on the left side the arch of the aorta lies over the root and the descending thoracic aorta passes behind it.

It is to be remembered that the important constituents of each pulmonary root are so arranged that on both sides the bronchus is in front, the pulmonary veins posterior, and the artery between bronchus and veins. The distribution from above downwards, however, is not the same on both sides, for whereas on the right side the bronchus is highest and the artery intermediate, on the left the artery occupies the highest level and the bronchus is next it. On both sides the veins are lowest.

The structure of the trachea and its branches, although showing a general plan throughout, varies greatly in detail in the different parts of the system.

The trachea is a firm, elastic tube lined by a ciliated mucous membrane, consisting roughly of five layers: the epithelial lining, the basement membrane, the inner fibrous coat, the muscular layer, and the outer fibrous coat. The three first-named constitute what is commonly known as the mucous membrane.

The epithelium may be regarded as having three layers of cells. The most superficial consists of columnar ciliated cells, the intermediate of pyriform cells, and the deepest of germinal cells. Among these different forms are a certain number of goblet cells.

Beneath the epithelium is the basement membrane, of a tough homogeneous substance.

The inner fibrous coat is formed by firm reticular fibrous tissue containing numerous elastic fibres.

The muscular layer in the trachea consists of scattered bundles of muscular tissue. Distinct transverse bundles are connected with the ends of the incomplete cartilaginous rings, which they may be said, in a sense, to render complete. Longitudinal bundles are to be seen external to these.

The outer fibrous envelope of loose connective tissue contains the cartilages. These cartilages are arranged as transverse but incom-

plete hoops, with the unfinished portion placed posteriorly, and filled up as above mentioned.

The fibrous and muscular layers contain lymphatics and small mucous glands; the latter open on the surface of the mucous membrane.

The bronchi and larger bronchia differ from the trachea mainly in two particulars. The cartilages become irregularly arranged plates outside of the muscular layer, and the muscular fibres form a continuous coat.

The smaller bronchia have no cartilages, and the walls consist of the ciliated mucous membrane, invested by a muscular coat and a fibrous envelope containing much elastic tissue and still possessing mucous glands.

At the point where each of the bronchia divides in its lobule into bronchioles, the epithelial cells become cubical and lose their cilia; the muscular tissue becomes less abundant and forms rings instead of a continuous layer; the fibrous layers become thinner and the glands are no longer present. These anatomical facts explain the much greater severity of capillary bronchitis, as compared with the disease in the larger tubes.

The bronchioles, opening out into infundibula in the lobules, gradually approach the structural characters found in the alveoli, but as we are not concerned in this section with the anatomy of the lungs we need not follow the arrangement further.

The air passages are well supplied with blood-vessels. Those going to the walls of the trachea in its upper part come from the inferior thyroid, and its lower portions from the œsophageal and bronchial arteries.

The bronchi and their branches are supplied by the bronchial arteries, which were referred to previously.

It must not be forgotten that the blood-vessels of the pulmonary apparatus are drawn from two sources. While the bronchial arteries supply the bronchial tubes and peribronchial tissues, and are finally distributed over the pleuræ, the pulmonary artery by means of its branches forms the entire capillary network of the alveoli. This distribution is of the highest interest, as it furnishes the obvious reason for certain well-known clinical phenomena. So long as any morbid process is confined to the bronchial and peribronchial tissues, the bronchial capillaries alone are involved, and the left ventricle from which they receive their blood supply is quite equal to any strain thus caused. If, on the other hand, a lesion implicates the pulmonary alveoli, as in emphysema consecutive to bronchitis, the pulmonary circulation is disordered and the right side of the heart suffers.

The lymphatic vessels mentioned as lying in the fibrous layer of the walls terminate in the bronchial glands and the glands in the neck, according as they come from the bronchi and their branches or from the trachea.

The trachea receives its nerve supply from the recurrent laryngeal above, and directly from the vagus below; it also has some filaments from the sympathetic system. The bronchi and their branches are supplied by the pulmonary plexuses, formed by branches of the vagus, twigs from the intercostal nerves, and filaments from the second, third, and fourth thoracic ganglia of the sympathetic.

The important fact must not be overlooked that the mucous membrane is in a constant condition of repair by the continuous loss of the effete cell elements and their replacement by those of younger growth. The superficial ciliated epithelial cells are thrown off when their life history has come to an end, so that their activity has ceased, and they are replaced by other cells, arising by the division of the cells of the deep germinal layer. The elements so produced pass towards the surface from the basement membrane as pyriform cells and gradually develop into the ciliated cells.

It must further be borne in mind that the mucous corpuscles are partly produced by the throwing off by the mucous membrane of embryonic epithelial cells and partly arise within the mucous glands.

Physiology.

The most important function of the trachea and its branches is conduction to and from the lungs, so that fresh air, already warmed in the upper air passages, may be supplied to the lungs during inspiration, for diffusion with that already exhausted, while part of that which has become effete may be removed during expiration. The performance of this important though limited rôle is assisted by the structural arrangements which have been considered, and from the physiological standpoint but few words need be devoted to this aspect of the subject.

In the presence of the elastic layer of the tubes there is a purely mechanical device whereby they are capable of adaptation to varying lengths, such as may be demanded not only by movements of the neck but also by the expansion and contraction of the lungs. By means of the muscular fibres provision is made for a certain amount of regulation of the calibre of the tubes—an amount which varies in the different regions of the system. In the trachea the extent of this alteration in the lumen must be slight, but in the case of the smaller bronchia and in the bronchioles the size of the tubes may

undergo very considerable modifications; since in the former there are only scattered bundles of muscular tissue, while in the latter the muscular fibres form a complete ring.

By means of the mucous membrane the air passages have provision for keeping the surface of the main channel and its branches smooth, for removing any extraneous materials, and for maintaining communication with the receptive nervous centres.

The mucous secretion supplied by the cells already described serves a twofold purpose. It causes the internal surface of the tubes to be moist and smooth, so that the inflowing and outflowing tide of air may pass to and fro with the least resistance. But it also entangles and retains any foreign particles of matter, thus preparing for the second function of the mucous membrane.

The removal of any extraneous substances is effected by two processes, each of which is complementary to the other. The cilia with which the columnar cells are provided continuously sweep mucus upwards, so that the foreign particles are constantly driven toward the upper air passages where they are got rid of. As has been seen previously, the cilia are absent in the finer ramifications of the system, which fact, along with the smaller lumen, explains the much graver dangers attending upon inflammation of these capillary tubes, owing to the difficulty in the expulsion of inflammatory products. The act of coughing, by the forcible compression of the lung, propels the contents of the smaller bronchia as well as of the bronchioles upwards, and in the larger tubes the current of air produced by a cough acts directly upon any material contained in them, and carries it onwards in its upward course. By these two processes provision is made for the rejection of any adventitious substances, as well as for the removal of mucus that has served its purpose and has been thus rendered effete. It must not be forgotten, however, that the lymphatic vessels may absorb some of the impurities which obtain entrance into the respiratory tubes and carry them off, but when this takes place the foreign particles are deposited in the bronchial and cervical lymphatic glands.

The respiratory mucous membrane finally is endowed with a certain degree of sensibility. It is doubtful if the sensory nerves of the tracheal and bronchial mucous membrane have anything to do with the regulation of respiration, which function seems to be mostly dependent upon the condition of the blood circulating in the special respiratory centres. But the sensory nerves in question have their own special functions. When any foreign body comes in contact with the mucous membrane a sensory stimulus is at once transmitted upwards, and induces the motor impulse resulting in the expulsion of

the invading substance. The introduction of cold air into the bronchial tubes induces their contraction, no doubt through reflex action. Further, when the wall of the respiratory tube is in any way injured or diseased, sensations varying from slight uneasiness to actual pain are experienced.

Pathology.

In casting a rapid glance over the general morbid anatomy of the air passages, it seems natural to note in passing the abnormalities resulting from imperfect development. Such malformations are not uncommon in conditions of acephaly and analogous arrests of normal embryological processes. The trachea in such cases may be absent; it may communicate with the œsophagus; it may open externally upon the surface of the neck—the condition commonly termed tracheal fistula; diverticula may be present or septa may exist; the trachea in its whole length may persist as a double tube—the bronchi being as it were continued up to the larynx—and dilatations or contractions of the lumen may be found. The majority of these congenital defects are found in embryos which are not viable.

Various modifications in the distribution of the bronchi and bronchia are seen from time to time, and these are occasionally so marked as to be regarded in the light of malformations. Congenital changes of the lumen of these tubes may exist; narrowing of the calibre is at times present, and bronchiectasis has been described as a condition resulting from developmental defect.

Functional Disorders.

Spasm of the muscular coat of the smaller bronchia has long been believed to be the cause of the symptoms presented by asthma, and as will be fully explained in the section dealing with that disease it is the only theory which explains many of the phenomena. It is well known that the vagus has the power of regulating the calibre of the smaller bronchia, and Graham Brown[12] has shown that it probably contains fibres which can induce both contraction and expansion of these tubes. Biermer formulated the view that the spasm occurring in asthma has for cause a reflex stimulation of the vagus.

Passive Congestion.

As a consequence of circulatory disturbances passive congestion of the mucous membrane of the respiratory tubes frequently occurs.

As most of the bronchial venous vessels end in the superior vena cava and vena azygos, mitral lesions have not such an important influence over the bronchial as upon the alveolar mucous membrane,

but when the right heart becomes implicated the bronchial tubes must suffer. Certain pulmonary lesions, especially emphysema, may secondarily produce venous congestion, and pressure on the great veins may also be a cause. Collateral congestion is sometimes produced by disturbances of the circulation elsewhere, more particularly in the abdominal region. In such cases the mucous membrane is thickened and velvety; it has an excessive secretion, containing not only a large number of leucocytes but also many epithelial cells; there is often a great tendency to hemorrhage from the distended capillaries. On microscopic examination the inner fibrous coat is seen to be œdematous, there is distention of the vessels, and the basement membrane is thickened.

Active Hyperæmia.

Active hyperæmia often occurs in consequence of the inhalation of irritating substances in the form of dust or vapor. This condition is not frequently seen post mortem, but when it is observed it shows itself in the form of an intense injection of the mucous membrane with great increase of the secretion.

Tracheitis and Bronchitis.

Acute catarrhal inflammation of the respiratory tubes may chiefly affect the trachea and bronchi, the bronchia, or the bronchioles. In these three situations the affection is commonly termed respectively, tracheitis, bronchitis, and capillary bronchitis.

The essential anatomical changes in catarrhal inflammation of the tubes consist in hyperæmia of the inner fibrous coat, œdema of the basement membrane, and proliferation of the layer of columnar cells, as well as of more or less of the deeper germinal stratum, and of the epithelium lining the mucous glands. The result is thickening of the inner fibrous layer and deeper structures, with the presence of many leucocytes, denudation of the mucous membrane, and great increase in the quantity of mucus. The lining membrane presents a deep red color instead of the natural gray tint, and is covered with a mucous or muco-purulent secretion. At times the processes are so severe as to cause the formation of ulcers of the lining membrane.

The secretion is composed of the mucus poured out by the follicles and goblet cells, mixed with the embryonic epithelial cells.

Chronic catarrhal inflammation of the air passages may take its origin in repeated acute attacks, which lessen the vitality of the structures involved; but it very commonly rests upon some underlying

condition such as venous stasis produced by a valvular heart affection, or blood changes, *e.g.*, in lithæmia. It usually extends from the middle of the trachea to the smaller bronchia.

The essential structural changes are thickening of all the deeper strata of the walls, due to hyperæmia and œdema, with distention of the blood- and lymph-vessels. The basement membrane is also thickened, and the epithelial layers are almost entirely denuded. The appearance of the lining membrane is much altered. The color is usually of a deep purple shade, and the surface is smooth and glistening. It is composed of the basement membrane.

Acute croupous or fibrinous inflammation apparently begins by the development of structural changes similar to those seen in catarrhal inflammation, but after a short time a distinct fibrinous exudation is formed, more or less adherent to the mucous membrane. This is expectorated in favorable cases and presents casts of the tubes in which it has been formed. The casts are seldom entire in circumference when they come from the larger tubes, but they are complete in this respect when arising in the smaller tubes. They are hollow tubes when formed in the larger bronchia, but are solid when produced in the finer bronchi and bronchioles. These casts are pale in color, and show distinct layers when coming from the larger tubes. They frequently show an arborescent arrangement corresponding to the disposition of the branching tubes. Microscopically they are composed of a fine fibrinous network, in whose meshes many leucocytes are seen along with epithelial cells. Charcot-Leyden crystals and Curschmann's spirals are not uncommonly observed in the casts from the finer bronchia. The walls of the tubes are hyperæmic, swollen, and denuded to some extent; according to the age of the fibrinous deposit it may be closely adherent or free.

Chronic croupous or fibrinous inflammation sometimes follows catarrhal attacks. It is characterized by the same structural changes as are seen in the acute form, but is in every respect less intense.

Diphtheritic inflammation is almost invariably a sequel to laryngeal diphtheria. It consists essentially in the formation of a membrane composed of a fine fibrinous network, with many leucocytes and epithelial cells in its meshes, and the characteristic bacillus is found associated in varying proportions with other micro-organisms. The walls of the tubes are deeply injected and swollen, and contain great numbers of leucocytes.

Ulceration of the mucous membrane occurs as a consequence of many of the inflammatory conditions we have seen, but it also occurs from the deposition of granulation tissue in tuberculosis and syphilis, and from the pressure of malignant new formations.

Changes in the Lumen.

Narrowing and widening of the calibre of the tubes may be caused by many different conditions.

Stenosis may have its origin in foreign bodies which have obtained an entrance into the tubes; in new formations or contraction of cicatricial tissue arising in the walls; or in pressure from without caused by many lesions of diverse kinds. This narrowing may affect any zone of the respiratory tubular tract.

Foreign bodies within the lumen of the tubes cannot be included within the category of diseases falling within the scope of the physician, and they will therefore be left for discussion elsewhere.

Neoplasms.—In comparison with the larynx, the trachea and the lower air tubes are but rarely the seat of primary new formations. Tuberculosis, for example, is always secondary to laryngeal phthisis, associated with a similar change in the lungs, and is nearly always seen as ulceration. Occasionally an acute eruption of miliary tubercle is, however, seen in the tubes. Syphilis, on the other hand, may attack the trachea and bronchial tubes without the coexistence of any syphilitic disease of the larynx or lungs, and it is apt to cause stenosis of the tubes from the thickening of the walls and the growth of nodules, as well as from the development of ulcers with contraction of the resulting fibrous tissue.

Lymphoid deposits in the mucous membrane are found in leucocythæmia, and fibromata or lipomata may exist in the deeper layers. Enchondromata take their origin at times in the cartilages of the walls.

Among malignant forms of tumor both carcinoma and sarcoma at times occur as primary manifestations in the trachea and bronchial passages, but it is much more common to find these neoplasms invading the walls of the tubes as secondary changes, most frequently in consequence of the development of the process in the mediastinum, in the pleura, or in the œsophagus.

Compression.—External pressure upon the respiratory tubes may be caused by a great variety of different affections. Bronchocele and other changes in the thyroid gland, diseases of the lymphatic glands in the neck and mediastinal abscess, new formations in the œsophagus, and aneurysm of the aorta or of the great arterial trunks are the most frequent forms of disease involving the lumen of the tubes by external pressure.

Thickening of the walls, resulting in a certain degree of narrowing of the lumen of the tubes, is caused for the most part by syphilitic processes.

Dilatation of the bronchial tubes, or bronchiectasis, may affect one, or more commonly many, of the tubes. The most usual seat of the dilatation is in the second and third subdivisions of the tubes. It is not uncommon to find communications between adjacent dilatations.

The mucous membrane is usually somewhat thinner. It appears to the naked eye more delicate, and on microscopic examination the epithelial cells are found to be small and the cilia less distinct. The membrane is often seen in different parts to be hyperæmic and thickened, and in this case the inflamed patches show similar changes to those described under bronchitis. Fibrinous exudation is even sometimes observed. Ulceration of the mucous membrane is occasionally found in some part of the dilated tubes with loss of the epithelium, and occasionally changes even in the deeper layers.

The muscular and elastic coats become thinner and even the cartilages become smaller and sometimes entirely disappear. These structures in some parts, however, sometimes undergo thickening on account of inflammatory processes.

The formation of communications between adjacent dilatations appears to be produced by a process of absorption and atrophy. The neighboring dilatations become adherent and the atrophic processes lead to the formation of an opening by which the adjacent dilated tubes communicate with each other. The contents of the dilatations present very varying appearances, which will be fully described in dealing with the disease itself. The pulmonary tissue surrounding the dilatations undergoes in many cases considerable alteration. Consolidation, collapse, or gangrene may be developed, but on the other hand the surrounding tissue may remain almost normal.

Semeiology.

Among the symptoms produced by affections of the trachea and its branches are certain *subjective sensations*. Of these there are several varieties. A feeling of general tightness or constriction is not uncommonly experienced in the more chronic forms of disease, but it has no importance from the point of view of localization.

Varying degrees of uneasiness from feelings of slight rawness to sharp pain are often present, and while undoubtedly belonging to the class of referred or somatic sensations, they often form a very useful guide to the chief seat of the affection.

Dyspnœa is no doubt produced partly by deficient oxygenation of the blood, acting through the central nervous system, and also in part by the circulation of blood at a high temperature through these centres. It is not as a rule an attendant upon affections of the larger

air tubes, unless there is a considerable amount of constriction or a high degree of pyrexia; but in the smaller tubes, on account of the greater interference with the aëration of the blood, there is often severe dyspnœa. When there is obliteration of one of the bronchi there is great dyspnœa on account of the loss of function of one of the lungs.

Cough may be produced by irritation of any part of the tubular system, and is in itself absolutely destitute of any value as a means of distinguishing different forms of disease except in one form: the brassy cough of tracheal obstruction. Considered, however, along with the nature of the substances ejected, it gives much help in determining the locality and character of the affection present. Coughing is preceded by a very characteristic sensation of tickling at the back of the throat or above and behind the manubrium sterni, and this is the case whether the cough be induced by some local irritation, or by some reflex cause.

This is not the place to enter upon an examination of the physiology of coughing, but we may be allowed to recall the fact that the forcible act of expiration with closure of the glottis may have its origin in some direct irritation of the nerve centres, or in reflex causes whether connected with the respiratory mechanism or some distant organ.

The *character of the sputum* in many kinds of tracheal and bronchial affections gives useful indications. The expectoration in affections of the respiratory tubes necessarily differs, not merely on account of the varying portions which may be involved, but also with the different forms of disease which may be present. Of the many different forms certain varieties can be easily distinguished.

A tenacious, transparent sputum composed almost entirely of mucus, and containing a very small number of cells, is characteristic of the early stage of an acute catarrh. It is always viscid and tenacious, expelled with difficulty after severe coughing, and so adherent as to remain attached to a vessel, even when it is turned upside down. Such a sputum is usually colorless and transparent, but it may be grayish-white in tint and only semi-translucent. It very frequently contains a large number of air bubbles, caused by the severe coughing which produces its expulsion. A few streaks of blood are sometimes to be seen mixed up with the expectoration. On microscopic examination, this form of expectoration is found to contain very few cellular elements, which are for the most part mucous corpuscles and epithelial cells, usually of the ciliated columnar type, but not infrequently also of the squamous form.

A thick, opaque sputum is characteristic of the decline of the

hyperæmia and inflammation of early bronchial catarrh, and it is extremely common in chronic bronchial affections. This form of expectoration is by no means homogeneous, and in addition to the yellowish opaque secretion just mentioned there is also a certain amount of the viscid transparent secretion of early catarrh. The proportion of these in different types is variable, in regard both to the individual affected and to the stage reached. On microscopic examination such a sputum is found to contain large numbers of mucous and pus corpuscles, but there are also many squamous and cylindrical epithelial cells.

A dense, opaque sputum, characterized by a uniform grayish-yellow tint, is sometimes present, and is found on microscopic examination to be entirely composed of pus corpuscles. Such a sputum as this is rarely seen in affections of the tubes alone, and is usually found when some breaking down of the lung tissue is present.

Such are the commonest forms of expectoration found in affections of the respiratory tubes, but there are certain special types found in some forms of disease which will be detailed in the proper place. Of these the fetid sputum of putrid bronchitis and the characteristic expectoration of bronchiectasis are very marked.

The arborescent masses expectorated as pellets in fibrinous bronchitis affecting the smaller tubes, and the moulds thrown off by the larger ones in the same form of disease, are of pathognomonic significance.

Asthma is frequently attended by a sero-mucous sputum containing Curschmann's spirals and Charcot-Leyden crystals. In certain cases characteristic groups of cells point to the likelihood of a malignant process in some part of the tubular apparatus. Blood is frequently present in all types of disease and cannot be said to have any great clinical import. More particular descriptions of the different kinds of sputa will be found in the work of Troup.[13]

Certain *disturbances of the circulation* result from affections of the bronchial system. *Cyanosis*, produced by deficient aëration of the blood, is a common symptom in acute capillary bronchitis, on account of the great interference with the passage of the air through the small bronchia and bronchioles. But it is also present to a great extent in old-standing chronic bronchitis, not only because there is thickening of the walls of the tubes but also because there is more or less emphysema, produced by the long-continued expiratory strain, which lessens the aërating pulmonary area, and, as has been already said, directly implicates the pulmonary capillaries.

Closely connected with this subject are the *dilatation and hypertrophy of the right side of the heart* in chronic bronchitis. They owe

their origin to two circumstances, the organic changes in the capillary vessels, on the one hand, and, on the other, the mechanical hindrance to the blood stream in consequence of forced expiratory efforts. Right-sided dilatation reveals itself by unmistakable physical signs, which it is no part of our present duty to analyze. We may, however, very briefly refer to the well-known clinical fact that if the cardiac implication is of any standing, there may be seen pulsation or turgescence of the cervical veins, and there may even be passive venous congestion, giving rise to œdema, dropsy, and visceral catarrh.

Among the general symptoms of bronchial affections, *pyrexia* must be mentioned as an accompaniment of acute diseases of the mucous membrane. The degree of pyrexia is as a rule moderate in cases of inflammation of the larger tubes; but in the bronchitis of the smaller, more particularly in that of the capillary tubes, the temperature may rise to 103° or 104° F., and be accompanied by a great frequency of pulse and a still greater acceleration of respiration.

With regard, lastly, to the *physical signs* it is unnecessary in this introductory section to say much, as they will be described under the different diseases of the tubes. It may, however, be stated generally that inspection and auscultation reveal the most significant evidences of bronchial affections.

Therapeutics.

As in the treatment of diseases elsewhere, it is necessary to consider whether therapeutic measures may be remediable or only palliative. The methods employed must, for instance, be entirely different in the case of an acute bronchitis and in that of a malignant tumor of the root of the lung. In the former case remedial agents must be adopted with a view to restore to a healthy condition the mucous membrane of the bronchial tubes; in the latter palliative measures only can be employed in order to relieve the sufferer from the consequences of a fatal disease.

The general treatment of acute affections of the trachea and bronchial tubes must in all cases be conducted on the main lines necessary for all acute diseases as regards rest and diet, but there are many points with reference to which special attention is needful.

The *air* which the patient breathes may be modified in regard to its pressure, its temperature, its moisture, and its composition, and while an abundant supply of fresh air is called for in all cases, these four points must in every instance be carefully kept in view.

As regards the effects of atmospheric pressure, it is unnecessary to speak in detail, but it must be remembered that the rarefied atmos-

phere of a high altitude is apt to produce hyperæmia of the bronchial mucous membrane, while, on the other hand, the denser atmosphere of a low-lying district produces an opposite effect.

The temperature of the air requires careful regulation in all tracheal and bronchial affections, particularly in acute cases.

The moisture of the air may be modified to suit individual cases, and it must ever be borne in mind that warm, moist air has a remarkably soothing effect upon the bronchial mucous membrane.

The composition of the air may in a twofold way be defective and require modification. It may present too small an amount of oxygen, and it may along with this contain a number of waste products which retard the respiratory process. In many cases of serious interference with the access of air to the lungs oxygen may be given with much benefit.

In the later stages of acute catarrh the employment of compressed air is of real service. It acts by causing pressure upon the hyperæmic and turgid mucous membrane by means of which the access of blood is hindered and its exit is favored; there can be no doubt moreover that when there is a tendency to collapse, compressed air prevents the morbid process. The portable apparatus invented by Waldenburg [11] is that which is most suitable for general use.

This is the proper place to refer to the influence of *climate* in the treatment of bronchitis. Patients who are liable to bronchitis in the cold months should, if possible, pass these seasons in a warm climate, the particular locality in any individual case to be chosen so as to meet its requirements. The indications are to obtain such a climate as will be equable, free from cold winds, and favored by as much sunshine as possible. For Europeans the most suitable regions are the Western Riviera, Algiers, Madeira, the Canary Islands, and Egypt. For Americans many places may be recommended in North Carolina, Georgia, and other of the Southern United States, and in Southern California; or, if an insular climate seems advisable, the patient may be sent to Bermuda, the Bahamas, Barbados, or Jamaica.

A word may be added as regards *mineral springs*. In convalescence from acute catarrh and in chronic catarrh the employment of waters containing sodium chloride and bicarbonate with free carbonic acid is followed by gratifying results. Such springs are very numerous and may be recommended to patients who can afford to sojourn for a time from home.

With regard to *drugs*, there are very many which act upon the bronchial apparatus as regards its circulation, its secretion, its nervous mechanism, and its muscular structures.

The circulation in the vessels of the bronchial tubes may be stimulated by all means increasing the circulation of the blood throughout the entire system—cold, dry air; such vapors as ammonia and chlorine; the fumes of nitre; internal remedies such as strychnine, alcohol, ammonia, digitalis, and strophanthus, squills, and all aromatic oils. The bronchial circulation, on the other hand, may be lowered by all vascular and cardiac depressants, such as hot moist air, fixed alkalies, iodides, antimony, aconite, and ipecacuanha.

The bronchial secretions may be increased by alkalies, iodides, antimony, squills, ipecacuanha, senega, all aromatic volatile oils, resins, and balsams. The administration of warm fluids also increases the bronchial secretions.

Antiseptic expectorants are required in cases presenting fetid or putrid sputa. Such agents may be exhibited by the mouth, by intratracheal injection, by inhalation with steam, or by the use of a vaporizer. Turpentine, terebene, creosote, carbolic acid, menthol, eucalyptus oil, and many other drugs are active remedies of this class.

The sensory nerves of the bronchial mucous membrane are stimulated by cold, by all irritating vapors, by ipecacuanha, senega, and ammonia, while they are depressed by warmth and moisture, by the use of warm applications to the chest and of warm drinks, as well as by such drugs as ether, chloroform, chloral, and opium. The inhalation of menthol is also of much importance for this purpose.

The muscular structures of the bronchial tubes may be stimulated by most of the measures which excite the sensory nerves, and they may be depressed by those which lessen the activity of these nerves, but in addition there are certain special depressants which relax the muscular walls of the bronchial tubes, chief among which may be mentioned henbane, belladonna, stramonium, tobacco, lobelia, nitrite of amyl, and iodide of ethyl.

In the treatment of bronchial affections it is sometimes necessary to have the means of influencing the respiratory centre directly, and for this purpose strychnine, ammonia, and alcohol are most to be relied on. It may sometimes be necessary to diminish the excitability of the respiratory centre, and for such a purpose opium and its derivatives, chloral, and the bromides, may be adopted. In acute affections the most important indications are to relieve pain, to lessen dyspnœa, to avert cough, and to modify the expectoration.

The various *painful symptoms* connected with the bronchial tubes may be arrested by such remedies as act directly on the end organs of the sensory nerves, more particularly by the inhalation of warm medicated air. External hot applications to the chest, sinapisms, and the like unquestionably exert a powerful influence upon

such symptoms. Whenever it be possible it is well to avoid the employment of such drugs as opium, but in very many instances such remedies are imperatively demanded.

Dyspnœa demands an increased amount of oxygen supply to the blood, and the air must therefore be abundant, warm, moist, and contain sufficient oxygen. The position of the patient sometimes requires to be so arranged as to insure free action of the muscles of the thorax. When the dyspnœa is produced by spasm of the muscular coat of the smaller tubes, the direct depressants of the muscular structures of the bronchial tubes must be used, for example, lobelia, belladonna, and stramonium. For rapid effects nitrite of amyl and iodide of ethyl are to be relied upon, or nitre fumes may be employed. Sometimes it is necessary to give chloroform by inhalation. When dyspnœa is produced by secretions which are too profuse, the various expectorants must be given.

Sedative expectorants, by which phrase are denoted those drugs which increase the bronchial secretions while they at the same time lower the circulation, comprise the fixed alkalies and their alkaline salts, the iodides, antimony, and ipecacuanha. Such drugs are of great service in the early phase of acute catarrh, especially in sthenic patients.

Stimulant expectorants, or those which increase the activity of the circulation while augmenting the bronchial secretion, include alcohol, ammonia, squills, strychnine, senega, volatile oils, oleo-resins, and balsams. This class is of great value in the later stages of acute and in chronic bronchitis. The drugs just mentioned are also of especial use in cases of cardiac weakness.

Cough is nature's means for the removal of any irritant or obstruction, and it is sometimes not to be interfered with; but it is on the other hand occasionally useless, and even baneful, when produced by some cause which cannot be removed; such a cough must be kept in check. It is therefore obvious that the cause of cough must in the first place, if possible, be removed by means of expectorants and other drugs which act upon the secretory mucous membrane in cases susceptible of remedial treatment. Only when the cause of the cough cannot be averted are the various sedatives to be employed. All narcotic remedies without exception are powerful sedatives to the respiratory centre, and they may in this way stop the reflex act of coughing, produced by stimulation of this symptom. While the thoughtless employment of sedative remedies for the relief of cough must be condemned, it must nevertheless be allowed that their employment in suitable cases is but one part of a rational attempt to produce a return to normal conditions, as there is no doubt that the

act of coughing, when stimulated by any reflex cause, is apt to continue longer than is necessary to effect the purpose for which it was produced, and is liable therefore to continue or even to originate irritation in the respiratory tract. Irritative cough is therefore to be curbed.

Hemorrhage from the bronchial mucous membrane must be treated with reference to its causation. If it should take its origin in some malignant invasion of the bronchial apparatus, it must be treated on general principles. If, however, it is part of an inflammatory condition the treatment of that primary condition must be so modified as to meet the additional indication. It will usually be found that the external application of cold and the internal administration of ice are harmful or at best useless methods of procedure, and the inhalation of vaporized terebinthinates, along with a hypodermic injection of morphine, are the most certain hemostatics.

In *chronic affections* of the bronchial tubes the indications for treatment are in many respects somewhat different from those found in acute conditions. The great points to be borne in view are to restore the mucous membrane to a healthy condition, to stimulate the circulation, and to avert the evil effects of faulty aëration. The alkalies provide us with the most useful alteratives for modifying the mucous membrane, but in a very large proportion of cases suffering from chronic bronchitis as well as from bronchiectasis, antiseptic remedies administered by intratracheal injection, by inhalation, or even by intrapulmonary injection, are urgently wanted. The circulatory stimulants such as digitalis and strophanthus are often urgently indicated, while general stimulants along with tonic measures are required to obviate the results of the unhealthy state of the bronchial mucous membrane.

The employment of alkaline springs, and residence in suitable climates, must always be borne in mind for such patients as can take advantage of these methods of treatment. As chronic bronchitis varies so much in its characters and indications we shall not in this introductory section dwell at any length on its treatment. It can be more satisfactorily discussed in the section devoted to the disease.

DISEASES OF THE TRACHEA AND BRONCHI.

Primary affections of the trachea and bronchi seldom occur. When these parts are invaded by disease the morbid processes almost invariably take origin in extension from some adjacent lesion, or are but a part of some general condition. Inflammation and catarrh,

for example, usually extend downwards from the larynx, or upwards from the bronchial tubes. Carcinoma and sarcoma are almost always an expression of secondary invasion. In the vast majority of patients who present symptoms of tracheal and bronchial disease, the tracheal and bronchial affection from which they suffer is only one part of a larger group, of which it is but a subordinate member.

Inflammation.

Definition.—An acute or chronic disease of the trachea and bronchi:

Consisting in inflammation of the mucous membrane, which may be catarrhal, fibrinous, diphtheritic, or mycotic;

Caused by individual proclivity, climatic agencies, general infection, and microbic influences;

Characterized by local pain, cough, and expectoration, without symptoms of laryngeal or pulmonary disease;

Resulting in recovery in most of the varieties, but frequently in death in the diphtheritic form.

Varieties.

Catarrhal Tracheitis.

Simple catarrh of the walls of the trachea and bronchi, varying in degree from slight hyperæmia to profound inflammation, is an extremely common affection, linked on the one side with catarrh of the upper respiratory tract, and on the other with similar changes in the bronchial tubes.

Etiology.—Associated as it is with laryngitis on the one hand, and bronchitis on the other, inflammation of the trachea and bronchi owns the same causes as these affections. Among predisposing causes may be mentioned town residence, indoor occupations, luxurious habits, adult age, arthritic and tubercular diatheses, and previous attacks. Sex can hardly be said to have any influence. Of the exciting causes the most common are sudden alterations in the temperature of the air, the inhalation of irritating vapors, chills of the cutaneous surface, and the presence of certain acute diseases, more especially of whooping cough, measles, influenza, small-pox, and erysipelas.

Morbid Anatomy.—In simple catarrhal tracheitis there is merely a condition of hyperæmia, with swelling of the deeper structures, and increase of the mucous secretion. In the acute form the mucous membrane is of a deep red color, often with ecchymoses and excoriations; it is velvety or sodden, and is covered in parts with the adhe-

rent secretion, which is as a rule muco-purulent. In the chronic form the surface is purplish, bluish, or grayish; the membrane is swollen, the glands are enlarged so as to be easily visible, and superficial erosion is sometimes present. The secretion, more purulent than in the acute form, is spread over a larger surface.

Symptomatology.—The symptoms vary in gravity with the severity of the affection, and according as it involves the larynx or not. Catarrhal inflammation of the trachea and bronchi may be acute or chronic.

In *acute catarrhal tracheitis* there is local irritation varying from slight tickling to sharp pain, and this sensation serves in part to localize the affection. Attendant upon it is cough, at first dry and brassy, but afterwards soft, and accompanied by expectoration; the cough is usually more troublesome when the patient lies on the back as the posterior wall is the most sensitive part. This sputum is at first scanty, but afterwards becomes more abundant, although never so copious as in bronchitis. In the earlier stages the expectoration is mucous or sero-mucous in character—in the later stages it becomes muco-purulent or purulent; there is little, if any, dyspnœa. If the larynx is not involved there is no change in the voice. In some severe cases dysphagia is present. The constitutional symptoms depend on the severity of the disease. There may be pyrexia, with an elevated temperature and a frequent pulse, or there may be no febrile symptoms in the case of subacute forms of the malady.

Auscultation as a rule affords little aid in the determination of tracheitis, as the lumen of the tube is too large to be materially influenced by inflammation of the membrane or exudation on its surface.

The laryngoscope, in the hands of a skilled observer, gives great aid, for with a good light, and the glottis widely open in the phase of inspiration, the surface of the tracheal mucous membrane may without difficulty be seen. It shows a general diffuse deep red tint, with areas of a still more dusky hue, where the blood-vessels are deeply injected, and collections of the muco-purulent secretion adhere to the wall.

The examination of the trachea by means of the laryngoscope requires some modification of the methods of ordinary laryngoscopy. The patient must be placed on a level higher than that of the observer. He must be seated with the body and neck perfectly vertical, and his head must be bent slightly forwards in order to have the larynx and trachea in a line as nearly straight as is possible. From the relative position of observer and patient the light will be thrown upwards, and the mirror, which must be placed upon the

soft palate a little further forward than when the larynx is to be examined, must be held in a nearly horizontal position.

Chronic catarrhal inflammation often results from repeated acute attacks. It shows itself as a rule by means of a persistent feeling of rawness in the windpipe, attended by the coughing up of muco-purulent expectoration. There is little disturbance. This form of affection, like chronic bronchitis, is frequently associated with cardiac affections, and has chronic venous congestion as an etiological factor.

Auscultation is here again of no practical use. Laryngoscopic examination shows a dusky flushing of the interior of the trachea with patches of secretion.

Fibrinous Tracheitis.

The existence of such a disease as fibrinous tracheitis appears to be denied by many authors. It does, however, present itself as a distinct affection, quite unconnected with any diphtheritic condition, and characterized by a fibrinous exudation which produces a quasi-membranous deposit.

Etiology.—With regard to this subject little is known, but in some cases which have come under our own immediate observation there could be no doubt that there was an arthritic tendency, inasmuch as the patients had inherited irregular gout, showing itself in its protean manifestations.

Morbid Anatomy.—In croupous tracheitis the essential nature of the process is an inflammation characterized by the presence of a fibrinous exudation which clings to the mucous membrane and forms a stratified deposit.

Symptomatology.—*Acute* croupous tracheitis causes more intense subjective symptoms than are present in the simple catarrhal forms of inflammation, the cough is more harassing, and there may even be dyspnœa. There is also considerable pyrexia. The essential feature of this type of tracheal inflammation, however, is the nature of the sputum, which contains larger or smaller pieces of the fibrinous deposit moulded on the surface of the mucous membrane. Such pieces when cut in section and examined under the microscope are seen to be composed of several layers of a hyaline material, often containing leucocytes and hæmocytes between them; the sputum frequently contains streaks of blood.

On examining the trachea with the laryngoscope it might be expected that the false membrane could be detected. We are not aware that this has ever been described.

Chronic croupous inflammation of the trachea and bronchi is a somewhat rare and troublesome condition. It entails long-continued irritation, with cough and the expectoration of stratified fibrinous deposit; the casts thus voided are somewhat similar to those resulting from the acute form, but they contain more leucocytes and less blood.

Within recent years one of us has been consulted at intervals by a gentleman suffering from a recurrent form of chronic croupous inflammation. In his case any exposure was followed after a few days by symptoms of tracheal irritation, accompanied by the expectoration of a muco-purulent sputum, containing casts, varying in size, but always showing layers of different texture. These casts sometimes had a larger, sometimes a smaller number of cellular elements; each recurrence seemed to differ somewhat from those which had preceded.

Diphtheritic Tracheitis.

This form of inflammation is almost invariably a sequel to diphtheria, causing the usual throat appearances, but a few rare cases have been described in which the primary affection has been situated in the mucous membrane of the air passages. It is no part of our present purpose to discuss diphtheria in general, and we must restrict our attention to the disease as it is seen in the lower respiratory passages.

Etiology.—Sometimes the membrane is not continuous with that upon the throat, epiglottis, and aryteno-epiglottidean folds, and in such cases it is permissible to suppose that the mucous membrane of the lower passages has been inoculated by particles of secretion drawn inwards during the act of inspiration. In the vast majority of instances the membrane is continuous with that above, and it therefore undoubtedly owes its origin to extension. In the cases presenting a primary implication of the lower tract the contagion is probably conveyed directly to the local mucous membrane.

Morbid Anatomy.—The essential difference between the false membrane as developed in the tubes and that seen above the glottis is that in the former it is much less firmly adherent. It gradually thins as it descends, and in the trachea, or at least in the bronchi, it comes to an end and is continued as a muco-purulent deposit in the bronchial tubes. In a few cases, however, the membrane invades even the bronchial tubes. Bartels[15] pointed out that when this occurs the extension is mostly confined to those tubes which run towards the inferior and posterior portions of the lungs, and his observations have received universal acceptance.

Symptomatology.—Extension of diphtheria to the larynx and trachea does not as a rule occur until four or five days from the beginning of the disease. The symptoms which lead to the conclusion that an extension has taken place are that there is some dyspnœa from interference with respiration. The dyspnœa is of an inspiratory type attended by crowing or croupy sounds and a hard brassy cough. The breathing is frequent and forced, and on inspiration the supraclavicular and infraclavicular regions as well as the lower intercostal spaces are drawn in. This dyspnœa undergoes paroxysmal exacerbation from time to time. It is unfortunately almost impossible to make any useful employment of the laryngoscope in such cases, and the determination of the extension of the disease to the tubes can only be by the symptoms and physical signs of obstruction.

In those rare cases in which diphtheria primarily affects the trachea or bronchial tubes the pathological diagnosis must always be a matter of considerable difficulty. The local symptoms and physical signs are simply those of a bronchial attack, but the fact of exposure to infection, the evidence of depression, and the incidence of albuminuria may be helpful in diagnosis.

The examination of the sputum by modern bacteriological methods may determine the diphtheritic character of the disease by revealing the presence of the specific organisms in the sputum.

Mycotic Tracheitis.

This has been observed by Herterich,[16] and a similar condition has been described by Eichhorst.[17] The symptoms are persistent irritation with troublesome cough, and the expectoration contains characteristic organisms. Aspergillus and leptothrix have been seen, associated with micrococci and bacilli. On laryngoscopic examination the trachea is seen to have greenish or yellowish deposits with areas of hyperæmia. The condition is not in any way dangerous, but obstinately resists treatment.

Diagnosis.

The localization of inflammation in the trachea and bronchi depends in great part on exclusion. If the larynx be unaffected the voice is unchanged; if the bronchi and bronchia are not implicated there are no bronchial rhonchi. The sensations are of importance. If there be rawness or soreness over the windpipe, and especially if the sensations are aggravated on swallowing, drawing up the chin,

or turning the head to one side, the implication of the trachea is determined. Tenderness on pressure is also an important sign. Auscultation, as remarked above, is of little importance, but the examination of the trachea by means of the laryngoscope is of the highest value. Lastly, in the fibrinous and diphtheritic forms the nature of the expectoration is pathognomonic.

Prognosis.

In almost all cases of non-diphtheritic inflammation the prognosis is good, but it must not be forgotten that there is considerable likelihood of recurrence in all varieties, while the croupous form is much more difficult to remove. Further, it must be noted that in exanthematous, erysipelatous, and variolous inflammation the prognosis is much graver. Diphtheritic processes are at all times exceedingly serious, and in laryngeal and tracheal diphtheria the mortality is very high, not merely on account of the effects of the disease upon the respiratory processes, but also because of the tendency to cardiac failure.

Treatment.

The general treatment of inflammation of the trachea must be carried out on similar lines to that which will be given in detail when dealing with bronchitis. There are, however, some points of difference, mainly arising from the greater utility of local measures in the case of tracheal affections.

When there is a tendency toward the tracheal catarrh, prophylactic methods must be employed. While avoiding relaxing habits, the patient must be enjoined to keep the feet dry, the skin warm, and the digestive organs active. He must be taught to breathe through the nose, or even in bad weather to use a respirator. All irritating substances which could reach the trachea as vapor should be avoided and the habit of inhaling tobacco smoke must be strictly forbidden.

When an acute catarrhal attack is present the most successful mode of treatment is to act freely on the bowels with a saline aperient, and to administer some remedy which will cause diaphoresis, such as acetate of ammonia, along with ipecacuanha. At the same time if there be much uneasiness and cough a sedative must be employed, such as opium.

The application of a hot poultice over the lower part of the neck and upper part of the chest will be found most advantageous. But

of all means the inhalation of steam is most beneficial; its efficacy will be increased by the addition of some soothing agent such as benzoin or camphor or menthol.

If there is very scanty expectoration the use of the alkaline carbonates will be of service. If there is profuse secretion it will be well to administer terebene, pinol, or other gentle astringent.

In chronic catarrhal tracheitis the patient will require general tonic treatment. Iron, quinine, and strychnine may be used with advantage, and in many cases iodide of potassium will be found of singular service. Among local measures the application of astringent remedies is to be recommended. Eucalyptus oil, or pine oil, nebulized by means of an atomizer or nebulizer may be used. Chloride of ammonium, administered by means of any of the special inhalers, is also of signal benefit.

In the case of the fibrinous forms, the internal administration of alkalies, particularly of liquor potassæ, produces the greatest beneficial effect, but in certain instances the iodides seem to be even more satisfactory.

The local treatment must be carried out on lines similar to those above described for simple catarrhal tracheitis.

If there be any dyscrasic tendency, such as the gouty, it must be met by the use of specific remedies, and it need hardly be added that in the case of the acute zymotic affections each case must be treated on the general principles applicable to it.

In diphtheritic tracheitis the main indications are the same as those in primary diphtheria of the throat, but in addition special attention is necessary to the condition of the respiratory passages. This is not the place to dwell upon the general treatment of diphtheria, but it may be said that maintenance of the patient's strength, antiseptic applications to the primary lesion, and the administration of antitoxin are indicated. With regard to the employment of this last mentioned it must be said that its utility is a matter still *sub judice*, but from our observations we are strongly inclined to regard it as having been the means of bringing about recovery in some cases which appeared to be almost hopeless. In two or three instances in particular we have seen recovery after ejection of an entire cast of the trachea, bronchi, and bronchial tubes. In addition to the use of this remedy, inhalations of steam and of nebulized antiseptics must be employed. For mycotic inflammation inhalations of perchloride of mercury (1 in 1,000), carbolic acid, and iodine have proved of most use.

Ulceration.

Definition.—A disease of the trachea which is usually chronic but may be acute;

Consisting in destruction of the mucous membrane;

Caused by inflammatory, necrotic, or traumatic processes;

Characterized by obscure symptoms of irritation, explained in many instances by laryngoscopic examination;

Resulting frequently in obstruction or perforation, followed later by death.

Etiology.—We have seen that in simple tracheitis excoriation of the mucous membrane may occur, and there can be no doubt that the process may result in deep ulceration. It is, however, not common, and most ulcers of the trachea are produced by tubercular, syphilitic, or malignant infiltration. Traumatism, extension of adjacent inflammatory processes, or pressure upon the trachea from without are also causes of ulceration.

Pathology.—The morbid appearances found after death vary according to the nature of the process. In tubercular and syphilitic ulceration there is some thickening of the submucous tissues underlying the lesions, and in the malignant forms the entire wall of the trachea may be involved in the morbid process. In syphilitic and cancerous affections there is often much narrowing of the lumen of the trachea.

Symptomatology.—The clinical features are often obscure, but in most instances there is long-continued irritation with constant coughing. There is also very commonly to be noted the presence of some laryngeal affection or the existence of disease in other adjacent structures. Laryngoscopic examination under favorable circumstances reveals the erosions which are usually on the posterior wall. The ulcers may be few or many and present varying appearances. In syphilitic cases the edges are often rugged, the floor rough, and the outline irregular. When of tubercular origin the ulcers are usually smaller, less rugged, and more numerous. If the processes should cause narrowing, the symptoms of stenosis are present.

A very remarkable condition was observed by one of us in a case which died under the care of Professor Laycock in the Royal Infirmary in 1859. The patient, a man of about middle age, was admitted in a dying state. His breath had a gangrenous odor, and he suffered from the most distressing dyspnœa. Respiration was attended by loud sounds, combining the ordinary characters of coarse mucous râles with a peculiar stridor, such as the physician in charge had never heard before. The post-mortem examination furnished the

explanation of the stridor, and of his general symptoms, for it was found that, in addition to scattered patches of acute phthisis in the lungs, there was an abscess in the neck, deeply seated and communicating with the trachea by an opening about an inch in length. The edges of the aperture were gangrenous, and portions of five or six rings had disappeared, while the ends remaining were left bare, and protruded into the lumen of the windpipe. As no sufficient history could be obtained, it was uncertain whether the abscess was secondary to a tracheal change, or had arisen independently of any such condition and perforated the walls from without.

Diagnosis.—The diagnosis depends in part on the local symptoms, but also in part on the general condition of the patient. The nature of tubercular ulceration may be rendered clear by the discovery of the characteristic bacilli in the sputum—that of syphilitic changes by the absence of such organisms, as well as by the presence of other specific appearances.

Prognosis.—The outlook cannot in any form of ulceration be regarded as very favorable except in that which results from simple catarrhal inflammation, or in such cases as have their origin in a neighboring abscess which can easily be evacuated. In syphilitic cases the prospect is by no means so grave as in those owning a tubercular or malignant cause, yet even in them the danger of stenosis from contraction and infiltration is too great to allow the expression of other than a guarded prognosis. And it must never be overlooked that there is a possibility of perforation in every case of ulceration whether from without or from within.

Treatment.—Among local remedies we have to mention anodynes for the relief of pain and irritation, astringents to promote healthy action, and antiseptics to obviate the baneful activities of microorganisms. Certain drugs, such as carbolic acid, possess the power of fulfilling all three indications; others, as perchloride of mercury, can act only in two of the directions mentioned; others, like morphine, can subserve only one purpose. Speaking generally, the local use of menthol, eucalyptus, iodoform, and mercuric chloride will be found of greatest benefit, the two former being most serviceable in tubercular ulceration, the two latter in syphilitic cases. Morphine is often required in cases of malignant ulceration. All these remedies may be administered, dissolved in olive oil, by means of intratracheal injection, or still better in the form of a vapor produced by some good nebulizer, such as that of Dunlap.

While such are the local measures of most utility, the constitutional treatment must be carefully attended to by means of general remedies directed to the underlying diathetic tendency.

Stenosis.

Definition.—A chronic disease of the trachea or bronchi;

Consisting in narrowing of the lumen of the tubes;

Caused by extraneous substances within the trachea or bronchi, by thickening of their walls, or by pressure upon them from without;

Characterized by a sense of constriction with dyspnœa and stridor, often attended by cough and expectoration, and unaccompanied by laryngeal or pulmonary symptoms;

Resulting in recovery when the cause is eradicable, and in permanent obstruction in other cases; frequently in death when there is malignant disease.

Etiology.—Diminution of the lumen of the trachea and bronchi may be caused by bodies within them, by changes in their walls, or by pressure upon them from without.

Morbid Anatomy.—Among the first class are foreign bodies which have obtained an entrance into the air passages, the consideration of which belongs to the province of the surgeon, and new formations, which will be discussed shortly. Of those connected with the walls of the trachea the only causes which are at all frequent are brought about by cicatricial contraction or infiltration. Belonging to the third group are many lesions of the lower part of the neck and upper part of the thorax, which produce more or less pressure upon the trachea.

Symptomatology.—The general symptoms of stenosis are difficulty of breathing and characteristic alterations in the respiratory sounds as well as in the cough. There is very commonly a sense of constriction in the chest which may even be painful. The dyspnœa which is present is attendant on inspiration as well as expiration, but in most cases the inspiratory phase is that most interfered with.

The respiratory murmur is altered by the stenosis so that it becomes harsh and strident. It may be recognized without auscultation, as the sounds produced are often quite audible when the observer stands at some distance from the patient. Dr. Wyllie,[18] of Edinburgh, has compared the sound of the breathing to the growling of certain animals, more particularly of the leopard. On auscultating the chest the peculiar stridulous noise may be heard conducted over almost the whole thorax.

The cough is harsh and brassy and has been likened by Wyllie[18] to that of a gander. Sometimes it is attended by the expectoration of sputa from which diagnostic conclusions may be drawn.

The voice may be rendered weak by the lessening of the current

of air in consequence of the tracheal obstruction. This has nothing to do with the loss of voice produced by pressure on the laryngeal nerves, which is often present in the same cases.

One symptom is urged by Gerhardt [17] as of value in distinguishing between obstruction of the trachea or bronchi and stenosis of the larynx: In the latter case the larynx during inspiration makes a sudden and considerable movement of descent; in tracheal or bronchial stenosis this movement is almost absent.

Another point has been signalized by Demme,[20] who points out that, in long-continued cases of obstruction of the trachea and bronchi, the upper part of the chest becomes narrow.

The further symptoms, along with the diagnosis, prognosis, and treatment, must be considered under the special forms of obstruction.

Tumors of the Trachea.

Etiology.—Sometimes the onset of new formations of the trachea has been distinctly the sequel to long-continued irritation, but in a considerable number of cases on record there has been no previous condition which might be considered as in any way etiologically connected with the development of the tumor.

Morbid Anatomy.—Most of the tumors of the trachea are papillomata or fibromata, but by different observers sarcomata, carcinomata, and other forms of neoplasm have been described.

Symptomatology.—It may be said that during the earlier stages of the development of a tracheal tumor the clinical features remain for a considerable time latent, and it happens in some cases that the affection is first noticed when some additional malady, such as a catarrh, directs the patient's attention to the windpipe. The general symptoms are those of stenosis, but there are some points of a characteristic nature.

The symptoms, as above remarked, may for long be unimportant or even unrecognizable—indeed the lumen of the trachea may be greatly reduced before any definite symptom shows itself. There may be no cough and no expectoration, and when these are present they are often more the result of inflammatory changes which have followed upon or even been caused by the new formation. But a period arrives when the symptoms can no longer be overlooked or mistaken. The presence of narrowing of the trachea becomes evident from features to be later described, and sometimes from the character of the expectoration. Blood is often present, but is of no diagnostic import: there may, however, be shreds of tissue of such a character as to throw light on the nature of the obstruction.

In all cases the examination of the interior of the trachea by means of the laryngoscope should be attempted. In the hands of an expert this laryngoscopic examination of the trachea may yield valuable information, as the general appearance of the tumor and its position may be recognized with considerable accuracy.

Diagnosis.—The absence of any appearance of changes external to the trachea, and the determination by laryngoscopic examination of some body within its lumen are the points of most importance. It is by no means an easy matter, however, to make certain of the nature of a tumor. When the new formation can be seen its appearance may be significant. The papillomata are usually more distinctly nodular than the fibromata, and both of these are of slower growth and are not attended by such constitutional symptoms as are found in malignant tumors. They are also not accompanied by the expectoration of bloody mucus and shreds of tissue which are often present in some tumors.

Prognosis.—In all cases the prognosis is somewhat grave, as tumors which are structurally benignant may be from their position clinically malignant. The malignant neoplasms are always followed by unfavorable consequences, but the simple forms of tumor may be removed through the larynx or by tracheotomy.

Treatment.—The removal of tracheal tumors by the aid of the laryngoscope can be carried out only by those who devote themselves specially to laryngeal and tracheal diseases, and it would be beyond our province to enter upon details in this work. When any simple tumor is so situated that it is accessible by means of opening the trachea, there should be no hesitation in obtaining the services of the surgeon. If the tumor is malignant every circumstance must be taken into account before such an operation is decided upon. In such cases as will not permit of surgical interference, the symptoms may be palliated by means of sedatives, both in the form of inhalations and by internal administration.

Cicatricial Contraction.

Etiology.—Narrowing of the lumen of the windpipe from contraction of the walls is usually the result of tertiary syphilis.

Morbid Anatomy.—The essential change is a deposit of granulation tissue which develops into dense fibrous tissue but is often attended by caseation in parts.

Symptomatology.—The clinical features are simply those of stenosis in general, unattended as a rule by the presence of any characteristic expectoration. The examination of the trachea by the laryngoscope shows a narrowing often attended by ulceration of the surface.

Diagnosis. The absolute determination of the nature of the lesion can be made out only by means of the laryngoscope; and the specialist, by the combined use of the laryngeal mirror and tracheal catheter, may be able to come to definite conclusions in regard to the exact site and degree of the stricture.

Prognosis. The outlook cannot be regarded as satisfactory. The tendency of the process is to extend in every direction, and the risks of more or less complete occlusion are considerable.

Treatment. The successful management of such cases consists in the combined use of internal remedies and local methods. The one drug for syphilitic stricture is iodide of potassium, which must be given in heroic doses. From half a drachm to a drachm and a half, administered three times a day, may produce beneficial effects without much local interference, but usually the general treatment requires to be supplemented by topical applications or operative measures. The inhalation of iodoform is useful if there be any ulceration, while benzoin, creosote, and other soothing remedies may similarly be employed.

In some cases dilatation of the trachea has been carried out by means of the tracheal catheter under the guidance of the laryngoscope; often, however, tracheotomy is required, either below the stricture in order to admit a sufficient supply of air, or above it, in order to allow of its efficient dilatation, if it be situated far down.

Compression.

Etiology.—Many different lesions may produce compression of the trachea.

1. Changes in the thyroid gland. In simple cystic bronchocele there may be such a degree of pressure on the trachea as to produce symptoms of impending suffocation. It is to be remarked that it is by no means always the largest bronchoceles that cause the most serious symptoms. The situation of the goitre and the condition of the surrounding tissues have much to do with the effects produced. To Virchow[21] we are indebted for the first recognition of the interesting fact that when a goitre presses downwards behind the sternum it produces serious compression of the trachea. It is only right to add that this view is disputed by Ross.[22]

In Graves' disease there is very rarely any compression of the trachea, and there are consequently no symptoms of narrowing of the lumen.

2. Changes in the mediastinal glands. The mediastinal glands are not infrequently the seat of abscesses, usually septic or tubercular, and from these pressure on the trachea results. In the former

class the pressure is of brief duration, for one of two events is not long delayed: the patient succumbs to the malady, or the abscess discharges into some adjacent structure and the pressure is thus removed. Tubercular suppuration is much more commonly the cause of compression.

Malignant changes in the glands are by no means uncommon; they give rise to permanent compression.

Much less frequent in its occurrence is an invasion of the mediastinal glands by lymphoid tissue in leucocythæmia and lymphadenoma. Cases of this nature have, however, from time to time come under our observation and the lesion has produced the typical effects of tracheal or bronchial compression.

3. Changes in the vertebræ. Tubercular processes in the lower cervical or upper dorsal vertebræ produce abscesses in the retropharyngeal space, and when such purulent accumulations are of any size, pressure on the trachea results.

4. Changes in the œsophagus. Malignant disease of the gullet is not at all infrequent. If the seat of the disease be low down there may be a close simulation of the clinical features of aneurysm. It cannot be an uncommon experience for any hospital physician to have patients coming to consult him for a supposed aneurysm, which on examination is found to be a malignant affection of the œsophagus. When within the thorax the mass need not be of large size to cause considerable trouble by pressure.

5. Changes in the aorta. Aneurysm of the transverse portion of the arch of the aorta is perhaps the most common cause of compression of the windpipe. When an aneurysm arises from the ascending aorta it almost invariably projects outwards to the right, and the trachea is not implicated; but cases are on record in which such an aneurysm has taken an opposite direction and has caused tracheal pressure. Aneurysm of the descending aorta, if it causes pressure on any part of the respiratory tubes, acts especially on the left bronchus.

6. Changes in the heart. In cases of great effusion into the pericardial sac there is sometimes compression, and both King [23] and Friedreich [24] have described stenosis of the left bronchus caused by pressure of a dilated left auricle.

Morbid Anatomy.—It would obviously be out of place to enter upon the assemblage of morbid processes which go to make up the complex conditions just described, but the mode by which implication of the trachea occurs must be briefly considered. There is a marked difference in the effects on the trachea produced by different kinds of external agents. When the pressure is produced by a simple tumor

or by an aneurysm, the cartilaginous portions become softened and fatty and yield before the pressure. If the pressure is caused by an abscess, an inflammatory infiltration into the walls of the trachea occurs, with destructive results. In the case of malignant disease pressing on the trachea, there seems to be no doubt that the malignant process invades the walls of the trachea and causes thickening. It must not be forgotten that aneurysms and abscesses not infrequently rupture into the trachea and discharge their contents into its lumen, thus producing sudden death from suffocation.

Symptomatology.—The general symptoms of obstruction are present, and special symptoms point to the particular lesion which is the cause of the pressure. In bronchocele the obvious enlargement of the thyroid gland at once reveals the nature of the case, and it only remains to be determined whether the goitre has invaded the retro-sternal opening into the thorax.

When the obstruction is due to some affection of the mediastinal glands the case may present greater difficulties, and the determination of the cause may only be by inference. When there is undoubted obstruction, with fluctuating temperature, the cause will probably be tubercular change in the glands; with emaciation and cachexia, a malignant invasion; with special blood changes, leucocythæmia; and with characteristic glandular swellings elsewhere, lymphadenoma.

Retropharyngeal abscesses as a cause of obstruction are uncommon, but such cases are seen from time to time. When due to disease of bone, interference with the movements of the vertebral column often affords a hint as to the nature of the disease; in cases of simple suppuration of the cellular tissue we have to depend on other symptoms, such as stridor, and on careful investigation the abscess may be detected. The temperature and pulse in such cases are of much import.

When difficulty of swallowing precedes symptoms of tracheal obstruction the cause of the latter is almost invariably malignant disease of the œsophagus. In such cases rapid emaciation and its attendant symptoms will be found. Occasionally ulceration may produce an opening into the œsophagus, and distressing symptoms result from the passage of food into the air-passages; we have seen paralysis of both recurrent laryngeal nerves brought about by this process.

When aneurysm of the arch of the aorta produces compression of the trachea, it is accompanied by unmistakable phenomena: swelling and pulsation with dulness over the superior sternal area, systolic murmur, and accentuation of the aortic second sound; sometimes a change in the left radial pulse; frequently paralysis of the

left vocal cord; symptoms of pressure on the great veins and on the œsophagus; and in addition to the stridor and brassy cough, there is almost always the symptom of tracheal tugging first described by Oliver,[25] McDonnell,[26] and Ewart.[27]

Diagnosis.—In almost every case of stenosis from external pressure, the disease primarily present has existed so long that it has been recognized before compression is produced. The only difficulty is with regard to those cases in which there is compression from mediastinal conditions, and even here the attendant symptoms are usually so definite that there is little possibility of error. In every case the trachea should be examined with the laryngoscope when a bulging inwards may under favorable circumstances be detected.

Prognosis.—Except in the case of bronchocele and of simple tumors or abscesses situated above the thorax, the prognosis in tracheal compression is always grave.

Treatment.—The treatment of tracheal obstruction is that of the underlying disease, and on this head, therefore, little need be said, as the appropriate measures in each form are described elsewhere.

DISEASES OF THE BRONCHIAL TUBES.

Inflammation or Bronchitis.

History.—Bronchitis does not seem to have been recognized until the early part of the present century, before which time it was comprised under the general term catarrh or defluxion. The term bronchitis was introduced by Badham and Frank.

Definition.—A group of acute or chronic diseases of the bronchial tubes;

Consisting in inflammation of their mucous membrane, presenting the following varieties:

(*a*) An acute catarrhal process affecting the larger tubes; (*b*) an acute catarrhal process affecting the smaller and capillary tubes; (*c*) a chronic catarrhal process involving both larger and smaller tubes; (*d*) an acute fibrinous process, and (*e*) a chronic fibrinous process;

Caused by exposure to cold and wet, local irritation, congestions consequent upon cardiac disease, and various constitutional states, such as rheumatism, gout, and Bright's disease; it is most common in damp localities and during damp weather, in children and old people;

Characterized (*a*) in the acute catarrhal form affecting large tubes by pain behind the sternum, cough, some degree of fever, expectora-

tion (at first scanty, white, frothy, afterward more copious, mucous, and muco-purulent), by clearness on percussion, and by sonorous and sibilant râles on auscultation, without increase of vocal resonance;

(*b*) In the acute catarrhal form affecting smaller tubes, by similar symptoms, excepting that the pain is usually less marked, while the fever, dyspnœa, and tendency to asphyxia are much more prominent, and the râles are sibilant rather than sonorous and attended by fine crepitations;

(*c*) In the chronic catarrhal form by frequent attacks of cough and dyspnœa, with expectoration often of large quantities of frothy or muco-purulent fluid, by loud, sonorous, and sibilant râles, with coarse crepitations;

(*d*) In the acute fibrinous form by symptoms closely resembling those of the acute catarrhal variety, but accompanied by the expectoration of characteristic fibrinous casts;

(*e*) In the chronic fibrinous form by the ordinary features of the chronic catarrhal type, associated with the particular sputum;

Resulting, the acute forms in recovery or frequently in death (especially at the extremes of life); the chronic rarely in perfect recovery, and tending to recur in successive winters; each of the forms is apt to pass into the other and to induce, secondarily, other diseases, as collapse, lobular pneumonia, emphysema, and other changes.

Etiology.

Predisposing Causes.—Acute bronchitis is caused by a very large number of factors, some of which are personal to the individual and others due to external conditions.

Hereditary tendency is undeniably present in a considerable proportion of cases, not merely an inherited weakness of constitution, but even a special tendency to disease of the mucous membrane.

An individual predisposition is certainly in many instances present and seems to afford the only explanation why certain persons, as Biermer[6] says, with liability to nasal catarrh entirely escape bronchitis, and of the further observation of Walshe[7] that some individuals are more particularly liable to capillary bronchitis, while in others the larger bronchial tubes only are affected.

Bronchitis may occur at any age, but is more frequently found in the extremes of life, as is shown by the registrar-general's reports.

Sex does not appear to have any special influence over the number of cases of bronchitis as may be seen in the statistical tables, compiled from the returns of the registrar-general, which are contained in the well-known article by Roberts.[28]

Habits of luxury induce a weakness of the respiratory mucous

membrane, whereby it is rendered less able to resist external influences. Certain occupations render those employed in them more liable to bronchitis, more particularly such as involve exposure to sudden changes of temperature or to the inhalation of irritating substances in the form of dust or vapor.

Social position undeniably plays an important part in predisposing to bronchitis, which is mostly, for the reasons just stated, much more frequent among the poorer ranks of society.

The general health may tend to render patients more liable to bronchitis. A condition of simple debility, for instance, predisposes to this disease and certain constitutional affections such as tuberculosis, gout, rickets, Bright's disease, and other conditions, are still more likely to produce a tendency to bronchial catarrh.

The presence of any pulmonary disease is favorable to the development of bronchitis, and if the mucous membrane has previously suffered, it is rendered more liable to subsequent attacks. The susceptibility to the disease seems to become greater with each attack, and further, it is a common observation that certain persons suffer from acute bronchitis with each recurring winter.

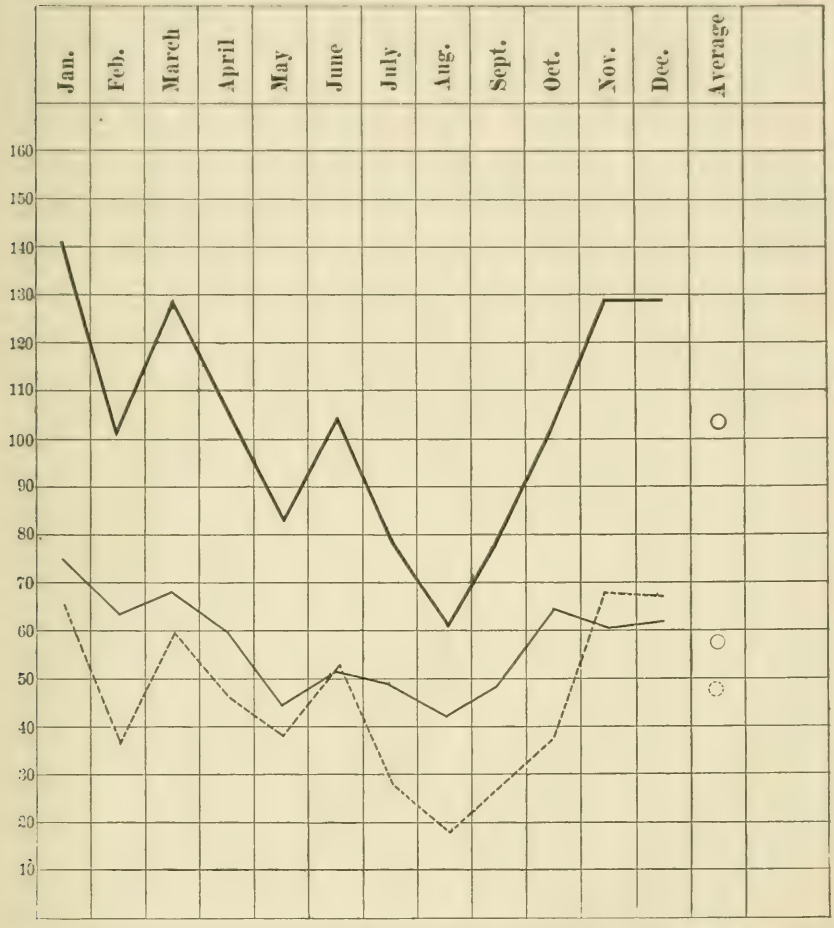
Any disturbance of the circulation which interferes with the return of blood from the bronchial veins has great influence in predisposing to bronchitis. Chronic heart diseases, therefore, more especially those which cause increased backward pressure on the great systemic veins, are to be regarded as potent causes of bronchial catarrh, in the way described in the introductory remarks.

Of external causes, climate holds one of the most important places. Bronchitis is much more common in climates which are cold and damp. It is, therefore, more frequent the further the distance from the tropics. Sudden and rapid variations in temperature, howsoever produced, exercise a notable influence over the incidence of bronchitis. Biermer holds that catarrhal affections of the chest occur largely when damp currents of air are blowing, and Lebert[9] accepts these views, believing moreover that sudden falls of temperature are frequently followed by outbreaks of bronchitis. Hirsch's observations[29] led him to conclude that the most important climatic condition is a low dew point, and that bronchitis occurs most frequently when the mean temperature and dew point have the smallest difference. The largest number of cases of bronchitis is met with during the colder seasons, from the end of autumn on to the early spring.

The statistics of the Royal Infirmary for the last five years have been collected for us by Dr. Lockhart Gillespie, medical registrar, and we present them in the accompanying chart and table, from which the varying numbers of monthly admissions can be seen. The fig-

ures, like those of other observers, prove that the disease is more common in the colder seasons.

Chart Showing Average Monthly Admissions for Five Years to the Royal Infirmary.



In the above chart the *acute cases* are shown by the *dotted line;* the *chronic* by the *light continuous line;* and the *total number* by the *heavy line.*

Admissions of Bronchitis to the Edinburgh Royal Infirmary, 1890–1894.

Year.	Acute Bronchitis.	Capillary Bronchitis.	Total.	Chronic Bronchitis.	Bronchial Asthma.	Total.	Grand Total.
1890	90	17	107	94	12	106	213
1891	120	36	156	109	13	122	278
1892	79	11	90	119	13	132	222
1893	69	27	96	144	14	158	254
1894	81	20	101	150	24	174	275
	439	111	550	616	76	692	1242

ADMISSIONS OF BRONCHITIS TO THE EDINBURGH ROYAL INFIRMARY, 1890–1894.—*Continued.*

	Acute Bronchitis.	Capillary Bronchitis.	Total.	Chronic Bronchitis.	Bronchial Asthma.	Total.	Grand Total.
January....	50	16	66	72	3	75	141
February...	30	8	38	59	5	64	102
March.....	52	8	60	62	6	68	128
April	39	8	47	53	7	60	107
May	29	10	39	42	2	44	83
June.....	40	13	53	44	8	52	105
July	23	5	28	43	7	50	78
August.....	14	4	18	35	8	43	61
September..	24	4	28	40	9	49	77
October	33	5	38	55	9	64	102
November ..	55	13	68	51	10	61	129
December ..	50	17	67	60	2	62	129
	439	111	550	616	76	692	1242

Exciting Causes.—In the great majority of cases acute bronchitis has for its immediate cause the influence of cold. This is seen more particularly in exposure to cold air on emerging from a warmer atmosphere; chilling of the upper part of the body during cold weather, more particularly with damp clothes; sleeping in damp beds, or some other mode of exposure to cold. In many instances the bronchial mucous membrane appears to be endowed with less resistance than the remainder of the body.

Other irritants besides cold induce attacks of bronchitis. Certain chemical vapors, such as chlorine or ammonia, for instance, are extremely irritating. The atmosphere may contain various forms of dust, such as flour, charcoal, or cotton, and the fog of large cities undoubtedly acts in this way.

Certain general diseases have a special proneness to produce bronchitis, apparently as part of the specific infection, such as measles, influenza, and other acute specific affections; and lastly, such morbid changes as tubercle may set up inflammation of the bronchial mucous membrane.

PATHOLOGY.

It is probable that the pathological explanation of bronchitis, when produced by cold, is that of paralysis of the blood-vessels affected by the sudden change in temperature, as explained by Rosenthal.[30]

The action of various forms of dust and of irritating vapors requires no explanation, but it may be observed that certain forms of matter in a fine state of division are much more noxious than others.

The inflammatory changes which occur in the course of some of

the acute diseases above mentioned are probably produced by changes in the blood. In some of the chronic affections, such as Bright's disease, impurity of the blood must, to a considerable extent, be the cause of the bronchial change.

The presence of such deposits as those of tuberculosis may be regarded as acting in a twofold way; in the first place, by the mere mechanical irritation of their presence, and in the second place, by the toxic effects which they produce.

Morbid Anatomy.

The anatomical changes which take place in bronchitis, the general features of which were briefly referred to in the introductory paragraphs of this article, vary within very wide limits, but the changes involved invariably consist of three essential processes—hyperæmia, swelling, and excessive secretion. The amount of hyperæmia varies from slight injection to deep congestion, and it may, as Biermer showed, affect either the superficial or the deeper structures or both of these layers. It is not to be forgotten that the hyperæmia may almost entirely disappear after death and leave the mucous membrane comparatively pale. The tint which is usually observed may be a bright red or dark purple, the latter more especially when the disease has been prolonged, or when there is any underlying affection interfering with the normal aëration of the blood. The mucous membrane is thickened, opaque, and velvety, the mucous glands become enlarged and distended, and in the smaller tubes the calibre is considerably diminished by these changes. Upon the surface of the mucous membrane are accumulations of secretion which differ from the expectoration by containing much more cylindrical epithelium than is found in the sputum. In the secretion found post mortem there are, however, numerous round cells, the secretions are sometimes clear, consisting mostly of mucus, or they may be highly purulent, and they not infrequently contain a certain amount of blood.

The bronchial lymphatic glands are almost invariably hyperæmic and enlarged. They sometimes contain extravasations of blood and pigmentary deposits.

The microscopic appearances chiefly show dilatation of the blood-vessels in the deeper layers of the bronchial mucous membrane with great increase in the deeper cellular layers, and shedding of the superficial layers of epithelium.

Secondary anatomical changes are produced in other parts of the respiratory apparatus. Diffuse bronchitis of the smaller tubes is attended by a certain amount of pulmonary hyperæmia and œdema,

and some broncho-pneumonia very frequently results. This, however, is not all, because the strain produced by coughing gives rise to some emphysema, while the inflammatory conditions produce on the other hand patches of collapse.

The pleura rarely suffers from any acute implication in the course of bronchitis, but it is probable that when bronchitis reaches the small tubes it may be a cause of pleuritic adhesions, which are otherwise difficult to explain in the case of many persons prone to suffer from bronchitis.

In bronchitis the heart is invariably more or less involved from interference with the passage of blood through the lung. The right side more particularly suffers from distention and repeated attacks of bronchitis produce permanent dilatation and hypertrophy. These changes in turn cause definite effects in the way of distention of the great veins with secondary changes in the internal organs and peripheral circulation.

Acute Catarrh of the Larger Tubes.

Symptomatology.

Acute bronchitis is usually ushered in by symptoms of catarrh of the upper air passages; but this is by no means invariably the case, more particularly in arthritic and tubercular patients, as well as in those who have suffered from previous bronchial attacks, in all of which classes the bronchial tubes may be directly and primarily attacked. As a rule, the invasion is somewhat gradual, but it sometimes occurs with extreme suddenness. The disease commonly begins with a sense of chilliness, not often, however, amounting to a rigor, and there is very frequently a feeling of soreness or aching in the body and limbs. Such symptoms are followed by the development of some pyrexia, the amount of which is usually slight, not often exceeding 100° F. The pulse is commonly more accelerated than the degree of temperature would lead the observer to expect. In children the pyrexia is much greater and the temperature may reach 103° or even 104° with a corresponding increase in the frequency of the pulse. In young children the initial symptoms may be extremely severe with the development of convulsions or with great nervous prostration.

A feeling of uneasiness behind the sternum, which may vary from a sense of rawness to acute pain, rapidly follows the initial symptoms, and this feeling may radiate throughout the chest. It is not at all uncommon for the patient to be able to point out which side is that mostly affected simply by the presence of such sensations.

At the commencement of an attack of bronchitis patients frequently complain of headache, undoubtedly due in most instances to the pyrexia, to the amount of which the pain is usually proportional.

Sleeplessness is a common occurrence, partly, no doubt, due to cough, but also in many instances produced by pyrexia. As the disease proceeds other nervous manifestations make their appearance, such as drowsiness, apathy, or even mild delirium.

In the first two or three days the surface is usually dry, but after this there is generally free secretion from the skin. There is no definite connection, however, between the disease and the amount of perspiration.

Hoarseness is commonly present, because in most cases of acute bronchitis the larynx is, at any rate to some extent, affected.

Cough commences early in the course of the disease. It is elicited by any cause exciting the mucous membrane or the nervous system; the act of speaking, a change of temperature, or any mental excitement will call forth a paroxysm of coughing.

The act of coughing is in the early stages of the disease unattended by any expectoration, and is therefore commonly spoken of as a dry cough. As the disease progresses the patient begins to expectorate at first clear frothy mucus, which is thin but tenacious and contains small whitish or grayish streaks; it may be tinged with blood or even be of a decidedly sanguineous character. On microscopic examination the paler streaks are found to consist of mucus and pus corpuscles. Sometimes a large quantity of this fluid is secreted, but in other instances it is much more scanty. After the lapse of from two days to two weeks the expectoration becomes opaque. The color changes to a yellowish or greenish hue, and it becomes much more viscid so that it adheres with considerable tenacity to any vessel. Under the microscope it is now seen to contain a very large number of pus corpuscles and epithelial cells from the bronchial tubes. The change from the earlier and later characters, as observed by Stokes,[5] is usually an accompaniment of a fall of temperature, and if a relapse should occur, the sputum usually becomes again clear and frothy.

Dyspnœa is almost invariably present in some degree, at any rate in the course of acute bronchitis, and the amount of breathlessness is directly proportional to the extent and severity of the changes in the bronchial tubes. The amount of breathlessness is, however, subject to a number of other conditions. The intensity of the pyrexia, as shown long ago by Ackermann,[31] produces a proportional increase in the number of respirations. The higher temperature of the blood circulating in the central nervous system, as has been proved by Fick,[32]

produces increased activity in the respiratory centre. The presence of any condition which interferes with the aëration of the blood also increases the breathlessness. A coexisting emphysema and œdema will markedly increase the amount of dyspnœa. It is more troublesome during the extremes of life—in children because their chest walls are more liable to yield, so that inspiration is impeded; in elderly persons because the elasticity of the respiratory organs is impaired and expiration is interfered with.

In severe dyspnœa the accessory muscles of respiration are employed. In the most severe forms orthopnœa is present. The patient may even be obliged to stoop forward and bend the head downwards. Spasmodic paroxysms of dyspnœa sometimes follow violent cough, and the breathlessness is always increased by any exertion.

Cyanosis is a characteristic feature in all serious cases of acute bronchitis produced by malaëration of the blood and closely linked with the preceding symptom. The tint varies considerably from a pale livid hue to a dusky flush, often associated with distention of the jugular veins and puffiness of the ankles. When the cyanosis is well marked there is considerable increase in the number of the red blood corpuscles, and it undergoes a characteristic increase during severe paroxysms of coughing.

Physical Signs of Acute Bronchitis.—In acute bronchitis the inspection of the chest shows that but slight changes are produced in its form, and the principal appearances are those attendant upon exaggerated respiratory movements in consequence of the dyspnœa which may be present.

In extensive bronchitis the chest is expanded in its upper part almost to its fullest extent, and it may be almost fixed in this position. The lower parts of the chest, more particularly at the sides, become often flattened or depressed. These effects are more particularly to be observed in children in whom the thoracic walls yield externally to pressure. If rickets be present, the distention of the upper part of the chest and the depression of the lower lateral regions produce a strong tendency to the development of the pigeon breast. If there be any collapse of the lung attendant upon the bronchitis, there may be local changes in form. When emphysema is produced, the spaces above and below the clavicles may bulge outwards. Sometimes, even in acute bronchitis, the apex beat of the heart may be displaced, and very commonly from depression of the abdominal viscera there may be a subcostal prominence.

Palpation of the chest cannot be held to furnish very important evidence in acute bronchitis. By the application of the hand, however, it is sometimes possible to detect some expiratory deficiency,

and this is more particularly the case in attacks of acute bronchitis occurring in those who have suffered from chronic forms of the disease. Unless there be some collapse, there is never any change in the vocal fremitus. Sometimes when there are considerable accumulations in the bronchial tubes rhonchial fremitus may be detected.

By percussion it may be ascertained in the majority of cases that there is little change. If there be, however, a very extensive bronchitis, the percussion sound becomes deepened so as more or less to approach the tympanitic character, more particularly over the upper and anterior parts of the chest. This clear percussion sound may be found to extend beyond the normal borders usually occupied by the lungs. If other processes be present, the nature of the percussion sound may undergo considerable change. Congestion of the bases of the lungs causes some diminution in the clearness of the percussion sound. If collapse takes place, a dull area of percussion sound will correspond to the extent of the collapse, and if lobular pneumonia be present, some muffling of the sound will follow.

By auscultation much more important physical signs are obtained. The character of the breath sounds is invariably modified, and although the respiratory murmur remains vesicular in type, it becomes harsh and exaggerated, no doubt partly on account of obstruction in the bronchial tubes, but probably also on account of the changes in the pulmonary parenchyma. Over some areas there may be weak breath, probably produced by total blocking of some of the tubes. The rhythm of the respiration undergoes characteristic changes. Both phases are lengthened, but expiration much more markedly, and the pause normally following the termination of expiration becomes shortened.

Bronchitis is always attended by certain accompaniments to the respiratory murmur, the most characteristic of which are rhonchi or dry sounds which depend on some obstruction of the tubes, swelling of the mucous membrane, accumulations of secretion, or spasm of the muscular coat. The pitch of these dry sounds is determined by two factors—the size of the tube in which they are produced, and the amount of obstruction which is present. In bronchitis confined to the larger tubes, low-toned or sonorous rhonchi are present and are best heard near the vertebral column posteriorly in the interscapular area. High-pitched or sibilant sounds are characteristic of bronchitis affecting the smaller tubes, and they are to be heard over the entire chest.

Moist sounds or râles are often present in bronchitis, and are usually large bubbling sounds. They seem to be produced by accumulations of fluid secretion, and are often audible in the early morning

before expectoration has taken place. If fine crepitations are present at the base, the fact speaks for the presence of congestion or œdema; if in the upper part of the chest, bronchial pneumonia is probably impending; if limited to one apex, the incidence of tubercle is to be feared.

Vocal resonance undergoes but little change in ordinary forms of acute bronchitis. If there be, however, much emphysemic tendency, it is diminished, and in cases with consolidation or collapse it is increased.

The general features of acute bronchitis may be considerably modified by any underlying diseased tendency; we may, for example, mention the bronchitis associated with influenza, in which the patient suffers from many symptoms connected with other systems. There is, for example, a marked tendency to cardiac failure shown by weak, irregular pulse and liability to faintness, or the digestive system may be profoundly disturbed and the absorption of food may be brought to a standstill. The kidneys may undergo changes and on the skin various rashes may show themselves, while profound nervous disturbances may be manifested. With regard to the symptoms connected with the chest, the important points are the severity of the breathlessness and the great tendency to extension of the disease. The inflammatory process is extremely likely to pass into the finer bronchial tubes and to invade the lobules of the lung, giving rise to a form of bronchopneumonia in which necrotic tendencies frequently occur. The special features, therefore, of influenzal bronchitis are the spreading character of the disease, the tendency to destructive changes, the liability to break down and to set up pneumothorax.

In rheumatic patients bronchitis sometimes occurs, and when this is the case the disease may be masked by the general symptoms of rheumatism. A high range of temperature, a very rapid pulse, profuse perspiration, and erythematous appearances upon the skin may be observed without any joint affection, but with symptoms of bronchial catarrh. Such cases appear to be worthy of the name "acute rheumatic bronchitis."

Arthritic patients may suffer with symptoms of bronchial catarrh, attended by many symptoms of acute gout; but it is without doubt much more common to find in patients who manifest the uric-acid diathesis a bronchitic attack apparently taking the place of a joint affection, and shown in part by some of the general symptoms of acute gout.

There is further a tubercular form of acute bronchitis in which, with profuse nocturnal perspirations and great wasting, there is a

tendency to bronchopneumonia, leading in its turn to destructive changes.

Course.

The course of the disease depends upon so many conditions that it is difficult to formulate any succinct statement on the subject.

The milder forms of the disease are attended by few severe symptoms, and the affection in such cases runs its course without much trouble. There may be little constitutional disturbance and the special symptoms and physical signs may disappear in a few days.

In elderly persons the disease is always more serious and involves considerable prostration. In children the inflammation is very prone to pass into the finest bronchial tubes, and to produce most serious symptoms.

The presence of any underlying affection is necessarily a complication involving much more serious risk, cardiac dilatation, Bright's disease, or any previous pulmonary affection rendering the patient much more susceptible to the influence of bronchitis.

Relapses often take place in acute bronchitis and necessarily give the disease a more serious aspect. Such relapses are more common in the extremes of life, and therefore very old and very young patients require more sedulous care in their management than adults. A relapse is much more likely to be attended by a soporose or even comatose condition, with a higher temperature, profuse perspiration, greater dyspnœa, and considerable prostration.

The duration of acute bronchitis has very wide limits. In the sthenic form of young adults the disease may pass away in a few days, but in the weakly, and more particularly when there is any underlying morbid tendency or any extraneous condition complicating the disease, its course be prolonged very considerably. On the other hand, in very severe attacks, the disease may cut life short within forty-eight hours, as in a case mentioned by Walshe.

Diagnosis.

The diagnosis of acute bronchitis does not as a rule present any difficulties. The presence of catarrhal symptoms with rhonchial sounds in the larger bronchial tubes, without any physical evidence of pulmonary disease, cannot be mistaken.

The presence of physical signs of bronchitis limited to the apices will naturally in all cases suggest the possibility of tubercular disease, and would lead the observer not merely to investigate the condition of the lung by percussion and auscultation with the greatest care, but would cause him to examine the expectoration from day to day

with a view to ascertaining whether tubercle bacilli or elastic tissue were present or not.

Acute general tuberculosis, when affecting the lung primarily, may at first be mistaken for bronchitis, because the general symptoms and physical signs are practically the same. But the degree of pyrexia and the amount of prostration are much greater in acute tuberculosis; profuse perspiration at night, and early delirium in the evening would point toward tuberculosis. A knowledge of the existence of tuberculosis in any other region would be of assistance, and the expectoration might afford evidence of the disease in the presence of tubercle bacilli.

The early stages of acute lobar pneumonia should never be mistaken for bronchitis, as the occurrence of a rigor, the sudden rise of temperature, the perversion of the pulse-respiration ratio, ought to be sufficiently distinctive before the physical signs of consolidation make their appearance. Not infrequently, however, do we meet with cases of pneumonia in which, until the development of these physical signs, the diagnosis must be regarded as doubtful.

The continued fevers are frequently attended by a certain degree of bronchitis, and the nature of the primary disease may to some extent be masked; but in these diseases the pyrexia, the prostration, and the more special symptoms should afford reliable evidence as to their nature. It must, however, be admitted that, more particularly in enteric fever, considerable difficulty may exist in regard to diagnosis.

In children the disease may be closely simulated by the early stage of whooping-cough, and the diagnosis may be impossible until the characteristic crowing inspiration has been developed. The first period of an attack of measles may also present a great similarity to bronchitis. The appearance of the rash, except in the case of morbilli sine morbillis, comes in due course to aid the diagnosis.

Prognosis.

Acute bronchitis in the extremes of life is a very fatal malady. But during the intervening periods, although it produces a vast amount of suffering, it cannot be considered as dangerous to life. The points which increase the risks in bronchitis are the occurrence of complications affecting the respiratory and circulatory systems. Perhaps before all emphysema ranks as most formidable. Long-standing phthisis even, when the morbid process has been arrested, has less effect. Cardiac complications also exert a most unfavorable influence, and the presence of albuminuria is of serious import. The secondary bronchitis of influenza, whooping-cough, and measles must

be regarded as graver than simple primary bronchitis, and in children the coexistence of rickets affects the prognosis unfavorably.

Treatment.

The treatment of acute bronchitis must be based upon the general considerations stated in the introduction as well as upon the special symptoms of the individual case. The treatment must further vary with the different phases of the disease. On the appearance of the earliest symptoms prompt measures should be adopted. A dose of phenazone or salol, along with the use of the hot bath, followed by diaphoretics and saline aperients, sometimes aborts the disease, more particularly if together with such remedies inhalations of steam containing sedative remedies be also administered freely. If there be much respiratory distress the application of poultices to the chest gives great relief, but they must be well made and frequently changed. Such measures will be rendered more effectual if the patient be kept absolutely in bed with a plentiful supply of pure warm air, and on a milk diet, especially milk and warm water.

The drugs which are most appropriate during this period are saline diaphoretics and ipecacuanha, which should be employed until the pyrexia has abated, and the expectoration has become quite free. At this stage some more stimulant expectorant may be employed, such as squills.

The aim of treatment in the early stage is to assist in reducing the catarrhal turgescence of the mucous membrane, and to hasten the natural result of the process as a free secretion. In an ordinary case of mild bronchitis such simple combinations as the following are useful:

℞ Liquoris potassæ, ... ℥ iij.
Spiritus chloroformi, ... ℥ i.
Infusi senegæ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every four hours.

Or,

℞ Ammonii carbonatis, ... ʒ i.
Ammonii bromidi, ... ʒ ij.
Vini ipecacuanhæ, ... ʒ iij.
Aquæ chloroformi, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every four hours.

In the early stages of the disease, if the patient is strong and of a well-marked sthenic type, there can be no doubt that antimony is a drug of great efficacy. It may be combined with ipecacuanha, a nitrite, or a simple alkali, due care being exercised in its administration. It must be discontinued if there be free expectoration with

low pulse-pressure and any symptoms of depression. The following forms of adminstration are useful:

℞ Vini antimonialis,
Vini ipecacuanhæ, ... āā ʒ iij.
Spiritus chloroformi, ... ℥ iss.
Liquoris ammonii citratis, . . . q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every six hours.

Or,

℞ Vini antimonialis, ... ʒ ij.
Spiritus ætheris nitrosi, ... ℥ ss.
Liquoris ammonii acetatis, . . . q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every six hours.

In patients manifesting an asthenic tendency, a stimulant line of treatment must be followed. In such cases carbonate of ammonium or the aromatic spirit of ammonia, along with cardiac stimulants, will afford the best results. The following formulæ are found to be very useful:

℞ Tincturæ strophanthi, ... ʒ i.
Spiritus ammoniæ aromatici, . . . ℥ i.
Aquæ camphoræ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every six hours.

Or,

℞ Ammonii carbonatis, ... ʒ i.
Infusi digitalis, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water every six hours.

In cases following upon influenza, nervine and cardiac stimulants, along with the free use of alcohol, are imperatively demanded. The prescription which follows has proved very beneficial:

℞ Tincturæ nucis vomicæ,
Tincturæ digitalis, ... āā ʒ iiss.
Tincturæ cardamomi compositæ, . . q.s. ad ℥ ij.
M. Sig. A teaspoonful in water four times a day.

Rheumatic bronchitis is best met by the use of the salicyl series of drugs, such as salicylate of sodium or salol, and in such patients the general treatment must be conducted on lines similar to those employed in acute rheumatism. The formula which follows is very serviceable in such conditions:

℞ Sodii salicylatis,
Potassii bicarbonatis, ... āā ʒ iij.
Syrupi tolutani, ... ℥ ss.
Aquæ anisi, ... q.s. ad ℥ vi
M. Sig. A tablespoonful in water four times a day.

Gouty bronchitis yields most readily to a combination of colchicum and alkalies, together with milk diet and aperient remedies. The following prescription can be confidently recommended in a sthenic case:

℞ Vini colchici,
Vini antimonialis, ... āā ʒ iij.
Potassii bicarbonatis, ... ℥ ss.
Aquæ destillatæ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water four times a day.

For asthmatic paroxysms the inhalation of chloroform, or of cocaine nebulized in a suitable apparatus, may be employed, and the following is very useful for internal use:

℞ Tincturæ lobeliæ æthereæ, ... ʒ iij.
Spiritus chloroformi, ... ℥ i.
Aquæ camphoræ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful every three or four hours.

If there be at any time intense dyspnœa it may be necessary to have recourse to inhalations of oxygen. In the administration of this gas some precautions are necessary. It must not be given in an undiluted form, but with free access of air, and it must be carefully warmed by passing through tubes lying in warm water. In such asthmatic or dyspnœic paroxysms, the application of a few leeches over the lateral parts of the chest may be extremely beneficial.

During the later stages of an ordinary catarrhal bronchitis the administration of alkalies and balsams proves most useful, such as:

℞ Ammonii carbonatis, ... ʒ i.
Potassii bicarbonatis, ... ʒ iij.
Syrupi tolutani, ... ℥ i.
Aquæ camphoræ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water three or four times a day.

Or the following:

℞ Tincturæ laricis (B.P.),
Tincturæ tolutanæ, ... āā ʒ iij.
Aquæ laurocerasi, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water three or four times a day.

We have found certain combinations, such as the following, useful for inhalation by means of Dunlap's globe nebulizer:

℞ Olei pini sylvestris,
Olei eucalypti,
Menthol, ... āā ʒ i.
Tincturæ benzoini compositæ, . . . ℥ ij.
Glycerini, ... q.s. ad ℥ iv.
M. Sig. For use with the nebulizer.

This is a useful remedy in simple bronchitis. If there be much irritation, the following acts like a charm:

℞ Cocainæ hydrochloratis, ... gr. xx.
Menthol, ... ʒ i.
Tincturæ benzoini compositæ, ... ℥ ij.
Glycerini, ... q.s. ad ℥ iv.
M. Sig. For use with the nebulizer.

In more advanced stages of the affection this may be employed:

℞ Creosoti,
Olei eucalypti, ... āā ʒ i.
Glycerini, ... ℥ ij.
Aquæ camphoræ, ... q.s. ad ℥ iv.
M. Sig. For use with the nebulizer.

If there be any fear of an underlying tendency to tubercle, it will be necessary from the first to make the patient inhale antiseptic remedies, either in steam or simply vaporized. The patient must further have food of the most nourishing kind that can be taken under the circumstances, and as soon as the acute stage has passed away he must be treated as if suffering from tubercular bronchopneumonia.

Acute Catarrh of the Smaller Tubes.

Capillary bronchitis, the name by which is commonly denoted an acute catarrhal inflammation of the small bronchial tubes and bronchioles, is not to be regarded as sharply marked off from the disease affecting the larger tubes, as numerous intermediate forms produce a gradation between them. It is not to be by any means regarded as in every case secondary to bronchitis of the larger tubes. It occurs not infrequently as a primary affection, particularly in the extremes of life; it further arises, independent of any affection of the larger tubes, in the acute infectious diseases. Not at all infrequently is it associated with bronchopneumonia, this latter affection being secondary to the capillary bronchitis.

Etiology.

With regard to the causation of capillary bronchitis, it is subject to the principles laid down in the previous section. Age plays an important part in the development of the disease; acute infectious diseases often precede it, and it is a common complication of many diathetic tendencies.

The morbid anatomy closely resembles that of bronchitis affecting the larger tubes, but the results of the inflammatory processes are

modified by the structural characters of the mucous membrane in the smaller tubes. These peculiarities have been referred to in the introductory section, and require no further remark in this place.

Symptoms.

The symptoms of capillary bronchitis are characterized by their severity and by the much greater danger attendant upon the disease. The pyrexia attains a considerably higher level, the pulse is so frequent that it is often almost uncountable, and its volume is frequently so small as to be almost imperceptible. There is commonly a high degree of cyanosis, the face becoming livid and the lips bluish-black, the tint of both becoming deeper during the paroxysms of coughing. The breathing is greatly interfered with. Severe dyspnœa, proportional to the obstruction in the air passages, is present in varying degrees, sometimes almost amounting to asphyxia. It is developed with extraordinary rapidity; the inspirations are short and hurried, the nostrils dilate with each, there is an abrupt jerky movement of the thorax, and the auxiliary muscles of respiration contract powerfully. The reason for the severe dyspnœa must be obvious from what has already been said in the introductory paragraphs. Not only is the lumen of the small bronchial tubes so narrow as to be readily obstructed by inflammatory products, but the epithelium lining these smaller tubes is not ciliated, and therefore, as previously mentioned, there is less provision for getting rid of the secretion. The amount of cough is very variable; sometimes it occurs only at infrequent intervals, but it may take place in severe paroxysms attended by great increase in the amount of cyanosis. One point of much importance is that the expectoration may be extremely scanty or even absent, only a small amount of extremely viscid sputum being expelled after severe spasmodic attacks of coughing.

The expectoration differs in some respects from that of ordinary bronchitis. It goes through the same stages, being at first seromucous and afterwards muco-purulent; but as it contains secretion from the smallest bronchial tubes which is not mixed with air, it has a tendency to sink in water. The sputum, therefore, is often seen to form a frothy layer upon the surface of water with trailing processes hanging down from the lower surface of the foam. Nervous symptoms are common. During the early stages there is much restlessness, more especially in the case of children, and the patient may toss about violently in bed. As the disease progresses there is a tendency to a lethargic condition and the patient lies in an apathetic state except when roused by the efforts to cough. Mild muttering delirium is an extremely common symptom towards evening.

but near the termination, in unfavorable cases, there is a great tendency to coma.

The *physical signs* of capillary bronchitis furnish numerous departures from those of the healthy chest. In adults, inspection may show no considerable deviation from a healthy standard except the marked dyspnœa shown by the exaggerated movements of the chest. But in children the thorax may show signs of more or less imperfect inspiration, more particularly when rachitis is also present. The lower part of the chest during inspiration is drawn in and the regions above the clavicle sink downwards, showing the difficulty there is in obtaining expansion of the lungs. Palpation confirms the evidence afforded by inspection and sometimes, though not commonly, it may in addition reveal the presence of rhonchial fremitus. The vocal fremitus undergoes little change, but it must be remembered that it is a sign of but slight importance in children. Sometimes, if there be no complications, percussion gives no evidence of dulness; but if there be collapse or bronchopneumonia, dulness may be present. Even when these complications exist they may be so deeply placed as to give no evidence on percussion. Sometimes the respiratory murmur is found on auscultation to be diminished, but it is sometimes harsher and rougher than in health. The respiratory murmur is in all cases prolonged and harsh. Throughout the chest there are numerous fine râles. If there are no complications there is no change in the vocal resonance.

Course.

The course of the disease in capillary bronchitis depends for the most part upon the presence or absence of complications. In uncomplicated cases, if the patient does not die within the first fortnight of the disease, recovery is the rule. In favorable cases when convalescence approaches, there is less dyspnœa and cyanosis, the pulse becomes less frequent in rate and of larger volume, the pyrexia diminishes, the tongue clears, the appetite returns, and on examination of the chest it will be found that there are fewer accompaniments. In fatal cases the dyspnœa usually deepens into asphyxia, followed by coma and death, but there are a great many symptoms which are shown during the evolution of the fatal termination. The pulse, for instance, may become more and more frequent, and the heart may fail. The temperature may rise to a high level and become uncontrollable, or the patient may be attacked by different nervous symptoms. Even in cases which recover from the primary affection, bronchopneumonia may follow, and either prove fatal directly or pave the way for tubercular infection to which the patient may even-

tually succumb; or emphysema may be developed along with collapse in certain portions of the lungs, and these diseases may be the cause of a fatal termination at a more distant period. Occasionally, after an apparently perfect recovery, the patient gradually sinks into a condition of marasmus, and, in spite of all endeavors to obviate the tendency to death, an unfavorable termination takes place.

Diagnosis.

The recognition of capillary bronchitis depends upon the amount of dyspnœa, the degree of cyanosis, the presence of high-pitched sibilant rhonchi, with fine crepitating râles, and the diagnosis as a rule presents little or no difficulty.

One symptom which may be found of importance is a retraction of the epigastrium and hypochondrium during inspiration. This retraction is very commonly seen in stenosis of the upper respiratory tubes, but such a condition is revealed by the stridor produced by the stenosis, and is little liable to be mistaken for the retraction seen in capillary bronchitis, where it is produced by an impervious condition of the bronchial tubes. A bulging of the supraclavicular and infraclavicular regions as first mentioned by Seitz, along with diminished movement in these regions, is also of some diagnostic import.

The greatest diagnostic difficulty is probably the distinction between capillary bronchitis and bronchopneumonia; indeed there can be no doubt that in a large number of cases no hard-and-fast line can be drawn between them because the dulness on percussion, which theoretically should be present in bronchopneumonia, is often wanting.

Prognosis.

The prognosis in capillary bronchitis is always grave and depends entirely upon the individual characteristics of the patient and the extent of the disease. Speaking generally, the more extreme the period of life the more serious is the outlook. The presence of any diathetic tendency and the existence of any previous disease must be regarded as greatly increasing the gravity of the malady. It is perhaps most fatal in young children who combine the tubercular with a pronounced rachitic tendency. When it attacks very old people the prognosis is rendered more gloomy when there is any arteriosclerosis or cardiac debility, and it must not be forgotten that, like all other acute pulmonary affections in old people, capillary bronchitis is apt to set up a fatal pyrexia.

Treatment.

The principles of treatment appropriate for bronchial affections in general, with regard to rest, air, warmth, and food must be observed, but there are a few points which demand particular notice.

With regard to food, for instance, it is well in every case, on account of the weakness of the digestive system, to restrict the patient entirely to peptonized milk and peptonized soup. Attention must be given to sleep, which must be procured by the use more especially of the bromides. This sleep, however, must not be allowed at any time to continue too long, and the patient should be roused up and fed every two hours, care being taken that he be moved from one side to the other at each awakening, so as to obviate any tendency to passive hyperæmia.

Alcohol must be administered freely in all cases, more particularly among the aged, remembering that for advanced life the adage is true that "wine is the milk of old age." In almost all cases some external application to the chest is necessary; a hot poultice produces beneficial effects, but if there be great prostration, it is still better to apply some stimulating liniment and wrap the chest up in cotton wadding.

As expectoration often does not take place in the course of capillary bronchitis, an emetic at the commencement of the disease, and repeated occasionally during its course, will be found beneficial. Ipecacuanha wine serves this purpose remarkably well, but in certain cases where there is no great weakness apomorphine may be administered hypodermically. In most cases small doses of ipecacuanha should be administered as an expectorant, and it is well to combine this drug with carbonate of ammonium or with the aromatic spirit of ammonia. If the circulation be embarrassed, strophanthus or digitalis must be employed. If dyspnœa be intense, threatening a fatal termination, the inhalation of oxygen will often be found to produce excellent results.

For an ordinary case of capillary bronchitis in a child we may recommend:

℞ Vini ipecacuanhæ, ... ♏ xl.
Spiritus ammoniæ aromatici, ... ʒ ss.
Aquæ anisi, ... q.s. ad ʒ ij.
M. Sig. A teaspoonful in water every four hours.

In cases where there is nervous prostration with much irritable cough:

℞ Codeinæ, ... gr. ℈.
Acidi hydrochlorici diluti, ... ♏ x.
Liquoris strychninæ hydrochloratis,* . . . ♏ xvi.
Syrupi tolutani, ... ʒ ij.
Aquæ, ... q.s. ad ℥ ij.
M. Sig. A teaspoonful in water every fourth hour.

Chronic Catarrhal Bronchitis.

Chronic bronchial catarrh varies greatly in its severity and in its characters. In its mildest forms it presents itself as a slight catarrh of the bronchial tubes during the colder seasons of the year. In more severe cases it is a permanent condition accompanied by great interference with the patient's general health and disturbance of almost every system of the body. It is further a frequent accompaniment of diathetic conditions and of circulatory and renal diseases.

Etiology.

The study of the causation of chronic catarrhal bronchitis involves a consideration of factors similar to those discussed in treating of bronchitis in general, but the causes differ somewhat in their relative frequency as compared with those of the acute disease.

With regard to causes affecting the individual, an hereditary tendency to the disease is more commonly found in chronic than in acute bronchitis, and the affection often occurs in a number of members of the same family. Chronic bronchitis is more common in elderly people, but it does not affect one sex more than the other. It is, like acute bronchitis, more common among persons who are exposed to sudden changes of temperature or to the inhalation of irritating substances. The existence of other diseases and diathetic conditions strongly predisposes to chronic bronchitis. Circulatory diseases which cause venous stasis produce great susceptibility to chronic catarrhal affections of the bronchial mucous membrane. Of external causes climatic influences are not so important as in acute bronchitis, for, as will be seen on referring to the table of admissions for bronchitis to the Edinburgh Royal Infirmary, at page 520, the numbers month by month vary much less than is the case in acute forms of the disease. The exciting causes of the disease are practically the same as those of acute bronchitis, but it is to be noted that any disturbance of the digestive processes or any interference with metabolism may arouse the latent tendency to chronic bronchitis.

* Of the British Pharmacopœia; contains about 1 per cent. strychnine.

Morbid Anatomy.

In an ordinary case of chronic bronchitis the appearances are usually extremely characteristic.

The mucous membrane of the tubes is very hyperæmic, of a deep purple hue, and with a smooth shining aspect. On closer examination, the surface will be seen to be in great part the basement membrane.

A cut section of the mucous membrane shows that it is considerably thickened throughout all its deeper layers, and on microscopic examination this thickening is seen to be entirely due to cellular infiltration. The muscular layer in great part disappears, and the glands and cartilages are also more or less destroyed.

Symptomatology.

The general symptoms in chronic bronchitis are by no means so severe as in acute forms of the disease, inasmuch as there is little disturbance of temperature or pulse. The alimentary system usually shows some interference with its processes. The tongue is commonly furred, the appetite is impaired, the functions of the bowels are capricious, the circulation always undergoes disturbance on account of implication of the right side of the heart, and there is therefore always a tendency to cyanosis and to general dropsy.

It is an interesting fact that when chronic bronchitis produces cyanosis there is a very considerable increase in the number of the red blood corpuscles which may be regarded as a compensatory process. If the circulatory disturbance is at all considerable, renal symptoms supervene, and albuminuria is not infrequently present. The nervous system is invariably subject to disturbance from malaëration of the blood, together with interference with sleep produced by cough.

The local symptoms in chronic bronchitis are analogous to those seen in the acute disease, but they vary within wide limits. There is as a rule much less sense of oppression in the chest, and the subjective sensations present seldom attain the proportions of actual pain. Breathlessness, however, is usually at least as severe as in the acute disease and it is necessarily much more prolonged. Cough is never absent, but it presents very different characters in different varieties of the disease, being at times accompanied by slight expectoration, at other times attended by profuse discharge.

The physical signs of chronic bronchitis differ but little from those of the acute disease, and the great distinctions between the two forms lie in the general symptoms. On examining the chest of a patient suffering from chronic bronchitis, unless the disease has

existed for a long time and produced secondary effects such as emphysema, there is but little to observe; but if the patient has suffered from the disease for any length of time, the chest will be seen to present the barrel shape characteristic of emphysema, with fulness of the supraclavicular spaces, rounding of the shoulders, and antero-posterior distention. Sometimes there are local flattenings of the chest produced by collapse in consequence of obstruction of the air tubes. On palpation, very commonly rhonchial fremitus may be detected. As a rule, the vocal fremitus undergoes no morbid change, but occasionally, and this is more particularly the case when circulatory complications exist, there may be an increase of the vocal fremitus in the lower part of the chest due to congestive processes. When the chronic bronchitis has been followed by emphysema, the vocal fremitus is diminished. On percussion, the sounds produced can scarcely be said to differ from those found in healthy conditions, unless there be on the one hand emphysema, which is expressed by a tympanitic character of the percussion sound, or any hyperæmic processes at the bases posteriorly, when there may be a slight degree of dulness. It must further be remembered that on account of the varying secretion within the tubes, the percussion sounds may differ somewhat from day to day. The breath sounds heard on auscultation undergo certain changes. In uncomplicated chronic bronchitis they become harsher with prolongation of the expiration, but the breath sounds are almost invariably so masked by adventitious accompaniments that their real character can with difficulty be determined. When it is possible to ascertain the character, it will invariably be found to be the puerile variety of vesicular breathing. The character of the breath sounds varies considerably in the different parts of the chest, and it is almost always impaired more or less from the constant association of chronic bronchitis with emphysema. The accompaniments which are heard are sonorous and sibilant rhonchi which vary in different parts of the chest, and which also differ from day to day, or even from hour to hour, in consequence of the alterations in the nature and character of the secretion. But in addition to these dry sounds some varieties of chronic bronchitis present moist sounds which may be small and crepitating, or may be large and bubbling, according to the size of the tubes.

In chronic bronchitis the sputum is extremely variable, both as regards its characters and its quantity. In ordinary forms of the disease the sputum is muco-purulent in type, but it differs greatly from time to time, being sometimes more fluid and frothy, at other times more dense and viscid.

These changing characters are due to the varying amount of local change in the bronchial mucous membrane at different times, from which it results that there may be a great increase in the serous constituents or in the cellular elements.

Varieties.

The features which have just been described are those of ordinary chronic bronchitis, but the disease presents some definite varieties to which reference must be made.

Dry Bronchitis.—This special variety of bronchitis was described by Laennec; its distinguishing features are severe paroxysmal cough with slight expectoration. This scanty secretion is usually viscid, tenacious, and of a grayish tint. The physical signs show that the chief seat of the lesion is in the smaller bronchial tubes. The spasmodic cough is often extremely severe, producing a cyanotic aspect and great exhaustion. It always results in some degree of emphysema and interference with the circulation. It is one of the most obstinate forms of chronic bronchial catarrh.

Moist Bronchitis.—Chronic bronchial catarrh is sometimes attended by such an excessive amount of expectoration as to merit the name of bronchorrhœa. This variety, as is commonly taught, resolves itself into two divisions—serous bronchorrhœa, and purulent bronchorrhœa. The former of these, described by Laennec as pituitous catarrh, is distinguished by a copious secretion consisting mostly of serum, and containing very few cellular elements. The sputum is frothy, with flakes of mucus; it is nearly transparent, and almost colorless. The physical signs show that the whole bronchial mucous membrane is involved. There is less tendency to implication of the circulation, and emphysema is not so constantly a sequel. Patients, moreover, in spite of the considerable drain caused by the excessive expectoration, do not emaciate to any great extent. Purulent bronchorrhœa is distinguished by the secretion of a large, often excessive, amount of expectoration, which is muco-purulent in type, the amount of cellular elements being at all times large, and sometimes so great as to render the sputum almost like ordinary pus. The physical signs show that the bronchial tubes, both large and medium-sized, are involved. One feature of this variety of bronchitis must be noticed, viz., a tendency to the development from time to time of pyrexia, which probably has its origin in the retention of pus within the tubes. There is not so much tendency to the development of emphysema in this as in dry bronchitis, and the circulation does not suffer to the same extent, but the patient's energies undergo considerable reduction, and emaciation results.

Putrid bronchitis is characterized by the fetid character of the sputum and of the breath of the patient. The characters of the sputum are pathognomonic. It is always copious and of the mucopurulent type, presenting a greenish-yellow color, and showing a disposition to separate out into three strata—an upper layer of grayish or greenish-yellow color, which is very frothy; a middle layer of transparent serum; and a lower layer, greenish or yellow in color, consisting mostly of cellular elements, and presenting very much the characters of ordinary pus. In this characteristic sputum there are small masses of a soft consistence and yellowish tint, the so-called plugs of Dittrich, which are composed of granular matter containing fatty globules with putrid animal substances and margaric and sebacic acids, together with some of the larger fungi. The odor of the sputum is fetid and penetrating, and, as is shown by the chemical examination, it depends upon the presence of decomposing processes, inasmuch as butyric and valerianic acids, along with trimethylamin, leucin, and tyrosin are found to be present.

The physical signs in no respect differ from those of ordinary bronchitis, and they show that the affection implicates the larger and the medium-sized bronchial tubes. From time to time pyrexia manifests itself, no doubt produced as in the case of purulent bronchorrhœa by the retention of decomposed material within the tubes. Patients affected by fetid bronchitis undergo considerable emaciation and loss of strength. Like bronchorrhœa it has not so much tendency to induce circulatory complications or emphysema as dry bronchitis.

Course.

In every kind of chronic catarrh of the bronchial tubes absolute recovery is rare. Every variety is apt to remain constant throughout the remainder of the patient's lifetime, irrespective of the season of the year, and to undergo exacerbation from time to time. Each has some tendency, but in varying degrees, to produce emphysema, dilatation and hypertrophy of the right ventricle of the heart, dropsical effusion into the serous cavities, œdema of the dependent parts, catarrhal processes in the alimentary tract, and albuminuria. Dyspnœa, cyanosis, loss of appetite, indigestion, dropsy, scanty albuminous urine, and many nervous symptoms reveal these disturbances.

Diagnosis.

The diagnosis of chronic bronchitis presents no particular difficulty in uncomplicated cases. The general and local symptoms, the

character of the sputum, the absence of dulness on percussion, and the characteristic adventitious sounds, almost inevitably lead to correct conclusions. The distinction between the different forms of chronic bronchitis can also be easily established from the appearances which have been described. The differential diagnosis of fetid bronchitis from bronchiectasis will be discussed under the latter heading, and the differentiation between fetid bronchitis and gangrene depends upon the absence or presence of broken-down lung tissue, as will be fully described in the section dealing with gangrene.

Prognosis.

The prognosis in chronic bronchitis is for the most part dependent upon the individual characters of each patient. As a general rule, when chronic bronchitis has once become established, it is apt to remain more or less permanent, and although it may not involve much danger to the patient, it greatly interferes with his usefulness. When chronic bronchial catarrh is associated with any dyscrasia its severity varies with the state of the diathesis, and when it is associated with any morbid changes in the circulation or in the kidneys, it is invariably more severe and more dangerous.

Treatment.

Chronic catarrhal bronchitis requires as many different lines of treatment as there are varieties of the disease, but there are some general principles applicable to all forms.

In the first place, the occupation of the patient must as far as possible be so modified as to lessen any interference with the respiratory functions, and a climate must be selected, if possible, which will tend to improve the condition of the mucous membrane.

In the next place, if any diathetic tendency is present, the treatment of the case must be so modified as to import into it the consideration of the indications furnished by any such tendency. If the bronchitis be linked with the uric-acid diathesis, for instance, the saline aperients, the fixed alkalies, and colchicum must be from time to time employed, and the functions of every eliminating channel must be carefully watched. If there be a tendency to tubercle, the use of cod-liver oil and iodide of iron, with residence at the seaside must be enjoined. If there be any manifestation of specific infection, heroic doses of iodide of potassium must be administered, and in all such diathetic cases the diet must be so regulated as to meet each individual indication.

It need hardly be mentioned that when there is any affection of the heart it must be met by proper treatment, and that if chronic

catarrh is associated with any change in the kidneys, such changes must be treated in addition to meeting the indications of the respiratory affection.

In every case of chronic bronchial catarrh the food should be as nourishing as possible, and yet of a nature to be easily digested and assimilated. Flatulence in particular must not be allowed, as any distention of the abdominal viscera interferes with the proper functions of the organs of the thorax, and therefore aggravates any respiratory disease. For the same reason the functions of the intestines must be carefully watched, and saline aperients will be found to be of inestimable value in almost all cases of chronic bronchial catarrh.

The clothing must be warm, so as to afford sufficient protection to the surface, and the patient must be prevented from any undue exposure, more particularly from draughts of cold air. If he cannot avoid a certain amount of exposure to cold air, he may be persuaded to adopt the use of a respirator.

In many cases of bronchial catarrh the use of stimulating liniments to the skin of the chest will be found very beneficial.

With regard to the special indications for medicinal remedies in different varieties of chronic bronchitis, it may be said that there are three main types of indications.

In dry bronchitis the internal use of alkalies is of importance in restoring the mucous membrane to a more healthy condition, and this method of treatment may be aided by the inhalation of an alkaline spray, administered by means of Dunlap's nebulizer—Ems water to which a small quantity of glycerin has been added is very useful in this way. If it be extremely difficult to expel the bronchial secretion, the inhalation of steam at night sometimes proves beneficial, and in a sthenic individual tartar emetic may be employed. In an asthenic individual, on the other hand, ipecacuanha will prove safer. The cough must be checked, and if possible it is well to do this without having recourse to opium or any of its derivatives. Spirit of chloroform and bromide of ammonium will in many cases be perfectly sufficient for this purpose.

In ordinary dry bronchitis we may give:

℞ Potassii iodidi,
Ammonii carbonatis, ... āā ʒ i.
Tincturæ camphoræ compositæ, . . . ℥ iss.
Infusi senegæ, ... q.s. ad ℥ vi.
M. Sig. A tablespoonful in water four times a day.

In moist bronchitis the drugs which are of greatest use are ammoniacum, turpentine, terebene, camphor, creosote, and balsam of

tolu, which may be not merely administered by the mouth but exhibited also by inhalation. Such a combination as the following may be employed:

℞ Tincturæ camphoræ compositæ,
Tincturæ tolutanæ, ... āā ʒ i.
Misturæ ammoniaci, ... q.s. ad ʒ vi.
M. Sig. A tablespoonful in water every four or six hours.

Or,

℞ Olei terebinthinæ, ... ʒ i.
Syrupi tolutani, ... ʒ i.
Misturæ amygdalæ, ... q.s. ad ʒ vi.
M. Sig. A tablespoonful in water every four or six hours.

In fetid bronchitis the stimulating antiseptic drugs are of most importance—carbolic acid, eucalyptus oil, sandalwood oil, myrtol, and menthol are of greatest efficacy. These may be administered by the mouth, they may be given by inhalation, or they may be applied by intratracheal injection. We may suggest:

℞ Olei eucalypti, ... ♏ xxiv.
Tincturæ camphoræ compositæ,
Spiritus chloroformi, ... āā ʒ i.
Misturæ ammoniaci, ... q.s. ad ʒ vi.
M. Sig. A tablespoonful in water three or four times a day.

Fibrinous Bronchitis.

This appears to be the best term for denoting a form of bronchitis in which there is the formation of a membranous deposit on the walls of the bronchial tubes. It is a somewhat uncommon disease, but in one or other of its forms it occurs from time to time in every kind of medical practice.

Etiology.

With regard to the causation of fibrinous bronchitis, almost nothing is known. It is more common in men than in women, and it has been found more particularly in early adult life. It is undeniably more frequent in those persons who have a tendency to catarrh, and a special predisposition has been observed in certain families. Most cases have been observed toward the end of spring. The disease appears to be absolutely unconnected with any diathetic tendency, although Waldenburg has seen it alternate with a skin rash of impetiginous type, and Biermer has seen it in tuberculosis.

Morbid Anatomy.

The fibrinous changes may be confined to certain districts of the bronchial system, or may be distributed over almost the entire arrangement of the tubes. The mucous membrane of the affected tubes may be hyperæmic and thickened, or it may be but little changed. The epithelium has sometimes been seen to be lost, at other times the epithelial covering was still present. The essential part of the morbid change is the formation of casts of the bronchial tubes, but these casts are sometimes absent on post-mortem examination, although numbers of them may have been present in the expectoration. When these casts are present, they present a stratified arrangement. Concentric layers lie one within the other, indicating almost with certainty that deposits of fibrin have occurred at different times. Such casts are occasionally very adherent, at other times they are easily removed. The consistence is usually found to be firm, but the casts of the finer tubes are somewhat softer. The color varies considerably, but it is usually yellowish-gray, occasionally tinged with blood. The peribronchial lung tissue sometimes remains unaltered, but in most cases some changes are present such as hyperæmia, consolidation, induration, emphysema, and collapse.

Varieties.

This membranous form of bronchitis is seen both in an acute and in a chronic form, of which the chronic is by far the most frequent.

The acute form of the disease sometimes presents itself in a recurrent fashion, and there is no very marked distinction between it and the chronic variety.

The essential feature of chronic fibrinous bronchitis is the recurrence of attacks at irregular periods, which may be spread over many years, as in the case described by Walshe, in which the disease lasted for more than fourteen years. In the chronic form of the disease there may be no rise of temperature, but occasionally a recurrence of the symptoms is preceded or accompanied by a rigor and some pyrexia. The pulse is often considerably accelerated, but this depends largely upon the disturbance of the circulation consecutive to the bronchial affection. There may be some disturbance of the digestive processes, even with some diminution of nutrition; occasionally the nervous system undergoes some irritative changes. The most important local symptom is severe dyspnœa with a hacking cough, followed by a characteristic expectoration.

Acute Fibrinous Bronchitis.

Symptomatology.—The general symptoms of acute fibrinous bronchitis differ from those of the ordinary catarrhal disease in the greater severity of the affection, and in the presence of a special type of expectoration. The disease usually begins like an ordinary catarrh with some pyrexia, cough, dyspnœa, anorexia, thirst, and general discomfort. The local symptoms either become gradually more violent with greater frequency and severity of the cough and a much higher degree of dyspnœa, or these graver symptoms may occur quite suddenly, and may involve much respiratory distress, breathlessness, and cough, and there may even be more or less hæmoptysis.

Sometimes there are no premonitory catarrhal appearances and the disease may set in with rigors, high fever, severe dyspnœa, and paroxysmal coughing. The dyspnœa and oppression constitute a very special feature of this form of the disease which is rarely, if ever, wanting. The cough is at first dry and brassy, and the voice never becomes hoarse, because the vocal cords are never affected in this form. The cough is followed in the early stages by little expectoration presenting the ordinary features of a catarrhal sputum, not infrequently showing traces of blood, but sooner or later the characteristic expectoration is present, and it consists in the expulsion of fibrinous masses. The expectoration is always preceded by severe efforts, and the discharge is at once followed by relief of the oppression and of the difficulty in breathing.

The fibrinous masses may fail to attract the attention of the patient or the physician inasmuch as they may be so small and so mixed up with mucus and pus as to escape recognition; but if the sputum is shaken up with water, the membranous deposit will be readily recognized.

The masses of fibrinous material are usually rolled up into more or less spherical forms, but when these are floated out in water they will be found to present different appearances, according to the locality in which they have taken their origin. When arising in the larger bronchial tubes, these masses seldom, if ever, present complete casts of the tubes but are seen in the form of larger or smaller pieces. When they are from the medium-sized and smaller tubes they form more or less complete casts, often presenting branching processes, corresponding to the distribution of the tubes. They are hollow in the case of the medium-sized and smaller tubes, but are completely solid when coming from the smallest bronchial tubes. Even to the

naked eye they present a stratified appearance, layers of a homogeneous translucent material being separated from each other by opaque strata, and they are often seen to be tinged with blood, which also is sometimes seen to be disposed in a stratified manner.

On microscopic section the homogeneous layers are found to be of a hyaline appearance, and the more opaque strata between them are found to be composed of masses of cells in which are very often a larger or smaller number of red blood corpuscles.

In acute fibrinous bronchitis the digestive processes are always considerably disturbed, and when the attacks are at all severe, symptoms of a soporose character are developed, usually causing characteristic appearances of faulty aëration of the blood, which may lead to a fatal termination. Improvement shows itself by a gradual diminution of the dyspnœa, the cough becomes less frequent and the amount of membranous deposit is diminished.

Upon physical examination of the chest no very characteristic phenomena are noticeable by which it is possible to recognize the affection. There is rarely any interference with the free movement of the chest, but, applying the hand to the surface of the thorax a rhonchial fremitus may be detected; there is never any change in the vocal fremitus, the clearness of the percussion sound remains absolutely unaltered, and although it is said that the respiratory murmur may be absent where large bronchial casts are firmly adherent, it is more usual to find that instead of being absent the breath sounds throughout the chest are harsh, the expiration is prolonged, there are dry sounds of very variable type, and there is absolutely no change in the vocal resonance.

Diagnosis.—The differentiation of this special form of bronchitis from the catarrhal type of the disease can be established only by means of an examination of the sputum. Casts of the bronchial tubes may be overlooked, as above said, in which case the real nature of the disease may not be ascertained. Such casts have been mistaken for coagulated blood, and from the presence of a certain quantity of blood adherent to the casts an error like this may easily be made, unless the sputum is examined with sedulous care. As soon as the expectoration of the characteristic membranous deposit takes place, the diagnosis becomes unmistakable, and so long as there are no casts the determination of the exact nature of the disease must remain uncertain. It is sometimes possible even before the appearance of the casts to determine the probability of fibrinous bronchitis, but it can never be a certainty, because the dry form of bronchitis may sometimes be marked by a very high degree of dyspnœa as we have already seen.

Prognosis.—In the acute disease, so far as previous observations can form the basis for generalization, a fatal termination has occurred in about half of the whole number of cases. The gravity of the prognosis will depend partly upon the severity of the general symptoms, and more particularly upon the ease or difficulty experienced in expelling the membranous masses. The age and strength of the patient exert considerable influence, and the presence of any diathetic tendency will render a case more unfavorable. Cyanosis and somnolence at an early period of the disease are of unfavorable import.

Treatment.—The main indications are in the first place to remove the deposit which has occurred, and in the second place to obviate the tendency to such deposits. Inhalations of steam are of undoubted utility, but of even greater use are inhalations, made by means of the nebulizer, of solutions of the alkaline carbonates containing glycerin; such methods are frequently quite sufficient. In more severe cases it may be necessary to administer an emetic, the best form being the internal administration of ipecacuanha, or the subcutaneous injection of apomorphine. The use of an emetic should be followed by a dose of calomel, and this followed by iodide of potassium, along with an alkaline carbonate. In order to prevent so far as possible future attacks, the patient should be recommended to follow out the general principles of prophylaxis against ordinary bronchial catarrh, and the employment of alkalies and iodides will be found of real use. Every remedy which will strengthen the general system is also of utility, and if there be any special constitutional indication such as the uric-acid diathesis or the tubercular tendency, it must be treated on general principles.

Chronic Fibrinous Bronchitis.

Chronic fibrinous bronchitis is as a rule seen as an addition to chronic catarrhal bronchitis, and it is sometimes rather difficult to say when the catarrhal merges into the fibrinous type. As a rule the symptoms become more severe from time to time, the breathlessness becomes more marked, the cough becomes more harassing, a cyanotic appearance is developed, and some hæmoptysis may even be present. After a severe paroxysm of coughing the nature of the affection is rendered clear by the expulsion of a fibrinous cast, after which all the symptoms are mitigated, until another cast is about to be ejected. But the symptoms are often much more severe than would be indicated by what has just been said. The dyspnœa may become intense, and excessive paroxysms of coughing may recur at very short intervals. The expectoration presents the same features as those which

have been described under the heading of acute fibrinous bronchitis, but here there is even more likelihood of the nature of the affection being misunderstood, from the fact that the characteristic deposits may be masked by mucus and by purulent expectoration. Occasionally slight febrile disturbances are present in this chronic form of the disease, and sometimes there is free perspiration, especially during the paroxysmal cough. Cyanosis and other symptoms of disturbed circulation are frequently seen, the nutrition of the patient undergoes greater change than in the acute disease, but there is not the same tendency to disturbance of the nervous system as in acute fibrinous bronchitis.

Physical examination as a rule fails to establish any difference between fibrinous and catarrhal bronchitis. There is no change in the form or expansion of the chest, there is no alteration of the vocal fremitus, there is no dulness on percussion, and although the respiratory murmur may be diminished in some regions connected with bronchi which have been closed by deposits, it is yet more common to find that the respiratory murmur is harsh and puerile with dry sounds of varying pitch, while the vocal resonance is absolutely unaltered. In very severe cases there may be diminution of the expansion of the chest when there is occlusion of the principal bronchial tubes.

Diagnosis.—The diagnosis, as we have seen in the acute form of the disease, depends entirely upon the presence of the characteristic membranous casts.

Prognosis.—The prognosis of chronic membranous bronchitis is much more favorable than that of the acute disease, and the vast proportion of cases affected make an excellent recovery. It must not be forgotten, however, that if emphysema be present, or if collapse of any part of the lung have taken place, or if there be such a diathetic tendency as tuberculosis, the outlook is correspondingly less favorable.

Treatment.—The treatment in the chronic form must be conducted on similar lines to those laid down for the acute disease. In addition to such methods of treatment, many of the remedies referred to in the section dealing with chronic catarrhal bronchitis are applicable here, and the conditions of the digestive, circulatory, and nervous systems require the most careful attention.

Bronchial Stenosis.

Definition.—A chronic affection of the bronchial tubes;

Consisting in obstruction to the passage of air along the tubes;

Caused by substances within the lumen of the bronchia, thickening of their walls, or pressure on them from without;

Characterized by more or less irritation shown by dyspnœa and cough, with peculiar stridor, best heard over the regions affected;

Resulting in most cases in permanent interference with respiratory processes, and in death when the cause is malignant.

Etiology.

Stenosis of the bronchial tubes is occasionally caused by some extraneous body, such as a pea, which has obtained entrance to the tubular apparatus. Sometimes it is produced by simple inflammatory thickening, but the most frequent cause of stenosis from changes in the walls is chronic syphilitic infiltration. Much more common than any of these factors, however, is external pressure, such as is found in carcinoma of the pulmonary glands or diffuse carcinoma of the lung, and in certain lymphoid deposits recurring, for example, in leucocythæmia.

Morbid Anatomy.

Examination of the lung and its bronchial tubes after death shows the presence of obstruction in some part of the latter and also reveals the cause of the narrowing. Sometimes this is a foreign body firmly impacted in a tube, with some inflammatory thickening of the tissues around. Very rarely indeed does a tumor arise within the tubes, but newly formed granulation tissue, especially in tertiary syphilis, sometimes causes thickening in certain parts, which by bulging inwards may simulate such a condition. Syphilis almost invariably gives rise to a widely diffused infiltration of the walls with a corresponding reduction of the calibre over an extensive area. Cancerous deposits may be localized in the pulmonary glands and produce pressure in limited regions; but more usually it is a diffuse infiltration of the lung spreading by the lymphatic channels and resulting in extensive compression. Leucocythæmia, through the lymphoid deposits formed in the pulmonary tissues, gives rise to analogous results. In all these different changes, however, the structural modifications are sufficiently characterized. It is not to be forgotten that areas of collapse are frequently produced.

Symptomatology.

The conditions falling under this category are by no means uncommon, but they are almost invariably secondary to other affections. The most noteworthy exception to this general proposition is found in the inflammatory thickening of the walls first described, so far as we are aware, by Andral. In the malignant, syphilitic, and leucocythæmic varieties the local change is but the result of general infection.

In consequence of this fact the bronchial symptoms usually rank as a phase of widespread disease. There are not merely the appearances classed as cachectic, but there are also the rapid wasting of malignant disease, the external ravages of syphilis, and the hæmatopoietic changes of leucocythæmia. And further, when there are deposits within the chest it is very common to find the bronchial symptoms somewhat masked by hydrothorax which results from the arrested return of blood caused by pressure on the pulmonary or bronchial veins. Such is the case frequently in carcinoma; such has been also observed by one of us in two or three instances of leucocythæmia.

The characteristic clinical features of stenosis of the bronchia are more or less dyspnœa, some cough with or without expectoration, and a stridor which may be heard over a larger or smaller area of the lung according to the extent of the obstruction. The stridor has a higher pitch than that heard in stenosis of the bronchi, and it may sometimes be found susceptible of analysis into two or more distinct tones blending into one, when several bronchia are implicated. The further symptoms and signs depend on the attendant conditions. If there be collapse an area of dulness will result with disappearance of the strident respiration to a greater or less extent, and comparative absence of breath sounds.

Diagnosis.

There is but little difficulty in determining the pressure of constriction of some part of the respiratory tubes, although it is by no means easy in many cases to distinguish between stenosis of the trachea and bronchi on the one hand, and of the bronchia on the other. But the less degree of disturbance, the more limited area over which the characteristic stridor is heard, and the higher pitch of the sound may serve to clear up a difficult problem.

Prognosis.

The outlook in all cases is serious, but it is not so grave in those which have a syphilitic causation.

Treatment.

In the rare cases of simple inflammatory thickening, the indications are practically the same as in chronic bronchitis. If it be clear that tertiary syphilis is present, appropriate treatment by massive doses of iodide of potassium must be employed. If, however, the obstruction is produced by malignant or lymphoid deposits, palliative remedies only can be used.

Bronchiectasis.

Definition.—A chronic disease of the bronchia, usually affecting many, sometimes only one branch;

Consisting in widening out of the cavities, with primary atrophy and subsequent thickening of the walls, usually associated with various changes in the contents of the tubes, the walls of the tubes, and the surrounding pulmonary tissue;

Caused by various conditions; but the most important form due to an atrophic process in the walls of the bronchi whereby they yield to the pressure to which the structures are naturally exposed. Most of the clinical features are due to secondary results of the dilatation, namely, the decomposition of retained secretion within the tubes, irritation of their walls, and of the surrounding pulmonary tissue by the products of decomposition, and the general constitutional disturbance from absorption of these products;

Characterized in some instances and stages by no special features, but usually, and always when well advanced, by the symptoms and physical signs of chronic bronchitis, with, in addition, a peculiar cough and expectoration, the physical signs of cavities, generally toward the lower parts of the lungs, and the symptoms and physical signs of the various associated morbid conditions of the pulmonary tissue and pleura. It is also attended by occasional attacks of fever with marked general malaise;

Resulting in most cases in prolonged suffering and ultimately in death by constitutional disturbance, by local pulmonary changes secondarily induced, or by complications affecting the lungs or other organs.

History.

In 1819 Laennec [3] published the first description of bronchiectasis, having himself become acquainted with the disease in 1808. He tells how his attention was drawn to it by a student of medicine who afterwards became Professor Cayol. Laennec described two chief forms, the regular and the irregular dilatations; taught that these might be associated with thickening, with normal thickness, or with thinning of the affected bronchial walls; and also showed that by communication formed between neighboring irregular dilatations, systems of cavities might arise which resembled multilocular tubercular excavations.

Laennec's views were perhaps incomplete in respect of the changes in the pulmonary tissue, and of the etiology of bronchiectasis. But

he was in our judgment more correct than many of his successors have been in his conclusions as to the relationship of the pulmonary tissue-changes to those of the bronchi; and he has had many followers in the belief that bronchial catarrh was the starting point, and stagnation of secretion the mechanical condition determining the dilatation.

Shortly afterwards Andral [30] published some original observations and suggested that changes in the nutrition of the bronchial walls and especially hypertrophic thickening must be taken into account, as well as the accumulation of mucus and the cough, in seeking an explanation of the malady. He recognized three forms of bronchiectasis, referring two of them, in which he said thickening of the bronchial walls exists, to hypertrophy of the walls, but he did not explain how such a change could lead to such a result. His third form (the moniliform or beaded) is, he said, usually accompanied by thinning of the walls and in it he thought that the dilatation may be a mechanical result of the distention of the tubes by mucus at the points where their parietes present least resistance, either from diminution of elasticity or from actual thinning.

Reynaud [31] published in 1835 some important observations upon obliteration of the bronchi. He found that dilatation was frequently associated with obliteration, sometimes on its proximal, sometimes on its distal side, and sometimes occurred in bronchi where there was no contraction. Thereby Reynaud came to the conclusion that those dilatations arose by way of complement.

Dr. Stokes,[5] of Dublin, writing in 1837, is the first British physician who deals with the topic in detail. His conclusions are:

1. That three classes are to be recognized:

(*a*) Cases with symptoms of chronic catarrh, with copious expectoration, existing for many (ten to fifty) years without constitutional symptoms of phthisis.

(*b*) Cases with symptoms of phthisis lasting five months to five or ten years—this principally in adults.

(*c*) Acute cases in children after whooping-cough.

2. That the affection occurs as an uncomplicated disease, or in conjunction with other lesions of which obliteration of the bronchi and tubercle are the most common.

3. That dilatation of the tubes may be accompanied by an atrophy of air cells, and thus the affected side of the chest be diminished in volume.

4. That in the same case we may observe predominance of dilatation of the tubes of one lung and of the air cells of another.

5. That continuous dilatation may affect a single tube without presenting any marked physical signs.

6. That numerous small dilatations may exist with symptoms of ordinary bronchitis.

7. That where continuous dilatation is decided and extensive, blowing respiration and extended resonance of the voice are heard.

8. When local dilatations are decided, the phenomena are those of suppurating cavities communicating with the tubes.

9. That although it is extremely difficult on account of the similarity of the physical signs, and in some cases of the symptoms, to distinguish this disease from phthisis, yet by observing the mode of combination and the succession of the signs, the rate of increase of the cavities, and the connection of these with the history of the case, we may in some instances at least arrive at a diagnosis which shall be correct.

10. That where a number of tubes are dilated in one lobe the case may be distinguished from tubercular anfractuosities by clearness of sound on percussion.

11. That if the case be seen early the fact of dulness not preceding the signs of a cavity may enable us to distinguish the disease from phthisis.

12. That so also the combination of extensive bronchial respiration with clearness of sound seems to be diagnostic of the dilated tubes.

With regard to the mode of production he concludes that most importance must be attached to loss of elasticity, contractility, and ciliary movement, and that the starting-point is in all cases bronchitis. As a result of inflammation, he believed that, first, the non-muscular longitudinal fibres lose their elasticity, and so cough and forced inspiration may dilate the tubes; second, that paralysis of the circular muscular fibres may occur, whereby the tubes dilate in the same way as the intestines do in cases of enteritis or ileus. Third, that the cilia of the epithelium lose their mobility.

Corrigan [35] in 1838, after describing and figuring the condition of the pulmonary tissue to which he applied the name cirrhosis, goes on to say that "the dilatation of the bronchial tubes is partly owing to the contractile process going on in the tissue of the lung—partly to the expansive action of the parietes of the chest in the act of inspiration," and he explains this contractile influence thus: "If there were but one bronchial tube with contracting fibrocellular tissue placed around it, then the contracting tissue would, as in the instance of stricture of the œsophagus or rectum, cause narrowing of the tube; but when there is, as in the lung, a number of bronchial tubes and the contracting tissue not placed around the tubes but occupying the intervals between the tubes, then the slow contraction of this tissue will tend to draw

the parietes of one tube toward the parietes of another, and necessarily dilate them." The dilatation is further favored by each expansion of the chest, which tends to draw open the tubes.

C. J. B. Williams,[36] writing in 1840, laid great stress upon the importance of the influence which is continuously exerted between the interior of the bronchial tree and the air—in inspiration by the air which enters to distend the tubes, in expiration by the tubes contracting to expel the air—and points out how, if the equality of the pressure be disturbed by more forcible pressure of the air on individual parts or on all parts, by loss of elasticity and contractility of the bronchial walls, dilatation of the tubes may occur. He also directed attention to the connection of cirrhosis of the lung with dilatation of bronchi and claimed priority to Corrigan in this observation.

Briquet[37] in 1841 drew attention to a variety of pulmonary gangrene attacking the walls of the bronchi and the surrounding lung tissue. His chief conclusions were that there is a form of dilatation of the bronchi in which the extremities of the tubes are dilated in sacs, which may be with or without dilatation of other parts of the bronchial tree, and that these dilated extremities are liable to gangrenous destruction independently of gangrene elsewhere. Dittrich and others have confirmed his observations.

Beau and Maissiat[38] in 1843 insisted on the dilating force exercised during the expiratory efforts of cough.

Fauvel[39] in 1844, in a valuable paper on suffocative bronchitis, related that in a considerable number of his cases he found a general or partial dilatation of the bronchial ramifications. Tolerably uniform in calibre, it was most marked in the finer tubes. It appeared to result from long-continued violent inspiratory efforts, in the course of which the air being hindered from passing further in its natural direction expanded the delicate bronchial walls.

Mendelssohn[40] argued that whereas under no circumstances can the inspiratory pressure exceed the atmospheric pressure, during the forced expiration which occurs in the act of coughing, on the other hand, when the vocal cords are closed the air is compressed and the pressure may be raised by one-seventh atmosphere.

Hasse,[41] in his "Pathological Anatomy," although agreeing with Laennec in regard to the forms he described, differed from him entirely and also from Andral as to the causation of the disease. "Bronchial dilatation," he says, "would appear to be engendered by bronchitis, by tubercular disease of the lungs, and by pleuro-pneumonia—the two former being specially prone to produce the spherical, the latter the cylindrical form." He agrees with Stokes in giving the first place to bronchitis as the antecedent and immediate cause of

bronchiectasis. Pleurisy of long standing may also bring about the condition by occasioning permanent collapse of a portion of the lung. Phthisis and bronchiectasis, he thinks, have a peculiar correlation: "The cure of phthisis is often brought about partly through the dilating of bronchial tubes—bronchiectasis determining in the organism certain conditions unfavorable to the further development of tubercle."

Barlow [42] gives an account of the morbid anatomy of a typical case of primary bronchiectasis—diagnosed as such during life.

Gairdner [43] suggested that all the so-called bronchial dilatations and all of those presenting the abrupt sacculated condition are in fact results of ulcerative excavations of the lungs (tubercular or other) communicating with bronchi, and expanded beyond their original size by the inspiratory force.

Dittrich,[44] writing in 1850, confirmed the observations of M. Briquet and others as to the occurrence of gangrenous inflammation in the walls of dilated bronchi and in the surrounding lung tissue. He also recorded the occurrence of septic metastasis, such as septic peritonitis, in cases of bronchiectasis. He was the first to describe among the contents of the cavities the occurrence of plugs and casts of the smaller bronchi. He believed that the coexistence of thin-walled and thick-walled bronchi in the same individual proved the accidental origin of these conditions, and their dependence on the amount of stretching or of inflammatory thickening they had undergone.

Rapp,[45] whose paper we have not had an opportunity of seeing, is described by Biermer as giving the first complete statement of opinion in Germany on the subject. It was communicated to the Medical Society of Würzburg in 1850. Biermer says that it contains much that is new but also much that is erroneous, particularly in the matter of histology. He pointed out that if catarrh could cause dilatation of the tubes, such a change ought to be much more frequent. He does not admit any direct connection between catarrh and dilatation, but associates it with pneumonia or pleurisy superadded to catarrhal irritation of the bronchi; and advances a very hypothetical explanation of the cavities formed after whooping-cough, viz., the tearing of the alveolar dissepiments by violent cough.

Virchow [46] contributed important observations upon the formation of cavities in the lungs and brought out the characteristic distinctions between tubercular and bronchiectatic spaces.

Cruveilhier,[47] while ascribing the condition to cough and the accumulation of secretion, emphasized the formation of intercommunication between neighboring dilatations.

Barth[58] thought that bronchiectasis may arise from changes (1) in the pleura, pleuritic adhesions drawing the bronchi open; (2) in the lungs, according to Corrigan's views; (3) in the bronchi, according to Stokes' view. These causes, singly or in conjunction, induce the results. He conceived further that an important influence is exerted by the pressure of the air, which being drawn forcibly in through the accumulated mucus does not pass out with expiration and so distends the tubes.

Lebert[59] concurred generally in the opinion of Stokes, but thought the loss of power dependent on nervous causes rather than upon inflammation of the bronchi.

Biermer[60] in 1860, while privat-docent at Würzburg, wrote an admirable paper in Virchow's *Archiv*, in which he assumed that we must accept as necessary conditions for bronchial dilatations:

1. Mechanical factors of dilatation. These may act either from within by intra-bronchial pressure, or from without by extra-bronchial traction.

2. Nutritive changes in the tissue of the respiratory apparatus which render the mechanical dilatation possible.

The mechanical relationships in question can be no other than the following:

1. Pressure upon the bronchial walls by stagnating secretion.

2. Increased air pressure within the bronchi. This may originate in the expiratory act of coughing.

3. Increased distention of the air passages during forced inspiration, which may act when the traction by the inspiratory muscles is, in consequence of disturbed mobility of the lungs, irregularly distributed on different portions of the apparatus, and permanently overcomes their power of resistance.

4. Extra-bronchial traction of the cirrhosed lung, whereby, apart from the respiratory pressure, the bronchial walls are opened out.

The nutritive changes which have been found connected with dilatation include:

(*a*) Pathological changes of the bronchial tubes—including not merely a loss of the vital properties of elasticity, contractility, and sensibility, but also narrowing and obliteration of individual branches.

(*b*) Changes in the alveolar parenchyma, directly or indirectly affecting the bronchi.

(*c*) Adhesion of the lobes of the lungs to one another and to neighboring parts, diminishing the mobility of the lungs.

Bamberger,[61] in 1864, made some contributions to the chemistry of the fetid sputum of bronchiectasis, giving the result of his examination of two cases.

Laycock [52] in 1865 connected fetid sputa with the rheumatic diathesis and conceived that when it exists there may be an excess in the pulmonary excretion of lactic acid, or of some other acid of the same class; or that the ordinary series of decompositions is intercepted and other products result in virtue of vital changes, whereby an irritant is brought into contact with the bronchial mucous membrane. If this be sufficiently powerful the same result will follow as with other powerful irritants (such as the vapor of iodine or chlorine), and thus a bronchitis with a copious muco-purulent discharge is excited. How easily lactic acid may undergo chemical changes is indicated by the fact that when lactates are left in contact with putrefying matter at a temperature of 86° to 96° F. butyric acid is produced.

Gintrac [53] related two very remarkable ways in which recovery has been observed to take place: (1) from the cretification of the contents, and the conversion of the walls into a sort of fibrous capsule; (2) from penetration of the pleura and thoracic parietes, and discharge of the contents outwards.

One of the present writers [54] in 1867 published in the *Edinburgh Medical Journal* a paper discussing bronchiectasis in its pathological and clinical aspects. He gave the facts of a number of cases which he had himself observed, and a résumé of the history of our knowledge of the disease up to the date at which he wrote. He referred to certain minor varieties of bronchiectasis, viz.,

1. A general dilatation of the bronchi from whooping-cough or from capillary bronchitis.

2. Dilatation from stricture of bronchi, first pointed out by M. Reynaud; this takes place on one or both sides of the stricture, and is manifestly connected with that condition.

3. Dilatations from long-standing indurations of lung substance, tubercular or inflammatory; these are always slight, local, and unimportant; they appear to result from retraction of the altered pulmonary tissue round them.

4. Spurious bronchiectases, which are in truth not bronchial at all, but the remains of chronic tubercular cavities or abscesses in the lung tissue communicating with bronchial tubes.

Having discussed these he claimed that two forms of bronchiectasis exist, viz., 1. The general or uniform. 2. The saccular or ampullary, and that the latter may be solitary or moniliform.

After describing the conditions of the walls, the contents, and the surrounding tissue, he discussed the various theories previously advanced and concluded that:

The essential element of bronchiectasis is a *primary atrophy* of the bronchial walls depending probably on constitutional peculiarities.

The bronchial walls being thereby weakened readily yield to the ordinary air pressure, and particularly to the increased pressure during acts of coughing—the process being thus analogous to that whereby a true aneurysm or a staphyloma of the sclerotic is formed. Accumulation of secretion in the dilatations necessarily results; and the changes in the walls and in the surrounding lung tissue as well as the constitutional symptoms are readily explained by the decomposition of the accumulated mucus. An account of the clinical history of the disease with illustrative cases concluded his paper.

Charlton Bastian,[55] writing in Reynolds' "System of Medicine," in 1871, devoted some attention to this subject. In addition to an account of the disease in its clinical and pathological aspects he summarized the various theories up to date, and from the diversity of these he concluded that condensation of lung tissue cannot in all cases be considered a necessary prelude to bronchiectasis. He pointed out that dilatation of the bronchi is not always present when cirrhosis of the lungs is fully established and when great contraction of the lung tissue has taken place. In one of his cases he confirmed Grainger Stewart's observation that fibroid change unmistakably advanced in a lung whose bronchi were already dilated. He agreed with the latter's views as the true explanation of cases in individuals who have had no long-continued cough and no bronchitis, and perhaps in some others. But in cases with chronic bronchitis he deemed it unnecessary to assume such a view, the theories of Williams and Stokes being sufficient to explain these. In cases due to cirrhosis, he thought the dilatation compensatory, to fill up spaces left by shrinking lung.

Wrany and Neureutter[56] in 1872 recorded a case in which there was a remarkable compensatory emphysema of the unaffected lung.

Gerhardt,[57] writing in the *Deutsches Archiv für klinische Medicin* in 1875, recorded two cases which are interesting in that he traces a relationship between bronchiectasis and rheumatic processes—attributing it to resorption of broken-down matter into the blood (a relationship which Laycock contended for, as regards fetid sputa, ten years previously). Gerhardt also gave details of his treatment by means of forcible compression of the chest to remove the purulent matter—great improvement following the adoption of this method after other means had failed.

Riegel,[10] in von Ziemssen's "Encyclopedia," gave an account of the disease and its morbid anatomy and histology. He thought "inveterate catarrh" the most important cause; he did not otherwise discuss the various theories.

Headlam Greenhow[58] in 1878 recorded a good case of the disease

which exhibited very clearly the physical signs of cavities without signs of accompanying consolidation. Greenhow believed bronchiectasis to be usually a secondary result of bronchitis but he admitted that Corrigan's theory holds good in some cases.

Leroy,[59] writing on the subject in 1879, gave as his opinion that the bronchia, especially their walls, are the seat of a primary alteration; that the lesion is one of nutrition in consequence of which the walls lose their contractile and elastic properties, and so readily yield to causes favoring dilatation. He believed the chronic pneumonia, referred to as an explanation, was really a consequence of the disease. He thus precisely confirmed Grainger Stewart's view, although apparently unacquainted with his writings.

Desplats,[60] also writing in 1879, recorded an interesting case of bronchiectatic cavities in the lung caused by compression of its main bronchus by an aortic aneurysm.

Theodore Williams,[61] in a series of lectures delivered in 1881 and recorded in the *British Medical Journal* for that year, gave an account of the disease in its clinical and pathological aspects. He refers to the folds and ridges of the mucous membrane and says that he has seen adjacent dilated tubes often freely communicating with one another. The walls of the bronchi are often so thin that the gray tissue of the lung can be seen through them. His account of the histology is that there is an infiltration of the walls by leucocytes, fibrous organization ultimately taking place, the non-striated muscle, glands, and cartilages disappearing, and being replaced by fibrous tissue until the bronchus equals a fibrous tube incapable of contraction. He considers that there are three principal causes, viz., (1) Inflammatory changes in the walls of the bronchi; (2) accumulation of secretion; (3) increased air pressure—chiefly inspiratory.

In 1886[62] Dr. Williams and Mr. Godlee recorded two cases where surgical interference was resorted to; they discuss the difficulties of diagnosing and localizing bronchiectatic cavities, and the indications for, and the arguments in favor of, operation; and Mr. Godlee gives details of his procedure.

Dr. Lauriston Shaw[63] in 1886 recorded the history and autopsy of a case of localized bronchiectasis caused by the invasion into a bronchus of a caseating bronchial gland.

In 1887 one of the present writers[64] recorded a case of unilateral bronchiectasis cured by operation and free drainage.

In the same year Barr,[65] of Liverpool, published the facts of a similar case, complete restoration to health following incision and drainage.

In 1888–89 two cases of bronchiectasis where operative treatment

was tried were put on record. Dr. Finlay's[67] case was not a favorable one, and empyema and hemorrhage followed the operation and hastened the fatal result. Dr. Mackay's[68] case was also very unfavorable for operative interference, being complicated by phthisis, and the patient died a month afterwards from pulmonary gangrene.

Hamilton[5] treats of the disease in his text-book of pathology. He accepts Corrigan's view as in the main correct, but incomplete; and he insists on pleural adhesions to a rigid chest wall as an additional factor.

Thin-walled varieties he thinks arise—

1. From increased expulsive efforts; *i.e.*, bronchiectasis is equivalent to an extension of emphysema to bronchi.

2. From accumulation of secretion.

3. From pulmonary collapse—*i.e.*, Williams' theory of concentrated atmospheric pressure.

In 1891[69] and 1892[70] Dr. Walter Carr wrote on the subject of bronchiectasis in children. He found a general history of measles and whooping-cough and chronic cough. As to the etiology, he thought that while several independent diseases may bring about the condition, it is a combination of them which as a rule produces it—and combinations are most frequently found in children.

Auld[71] wrote on the pathology of the condition in 1891. He describes the lesions pertaining to bronchiectasis as (1) atrophic, (2) inflammatory. He agrees with Grainger Stewart's description of the peculiar morbid anatomy; but his explanation of certain points is totally different; for example, he conceives that the bridge-like formations described arise by the fusion of the apices of papillary outgrowths of the mucous membrane. He lays great stress on the invariable persistence of the basement membrane in all the variety of changes which the mucous membrane may undergo, and he points out that that fact can always histologically distinguish a bronchiectatic cavity from a tubercular excavation, and that the same fact is alone sufficient to disprove Gairdner's theory. He in the end indorses Grainger Stewart's view that degenerative changes in the walls of the bronchi are the main factors in the production of the disease, and that the analogue of a bronchiectasis is a true aneurysm.

Wilson Fox,[72] in his treatise on "Diseases of the Lungs and Pleura," gave a very full account of the disease, and of the various opinions as to its pathology. He criticised the theories previously put forward, and argued from the variety of pulmonary conditions in which dilatation of bronchi is found that (1) no pathological changes are necessary for its production other than those which are found in the bronchi themselves, and (2) that the one condition common

to them all is an inflammatory change in the bronchial tubes, and that therefore (3) the dilatations are probably immediately caused by loss of elasticity and of muscular contractility. From a consideration of these facts he thinks it very probable that in cases weakened as above the dilatations are caused by the expiratory force exerted in cough. The rarity of the disease compared with bronchial inflammation he explains by the fact that the elasticity, and still more the contractility, of the bronchial muscles is not easily permanently destroyed.

Dr. G. H. Sutherland[73] in 1892 recorded an interesting case of bronchiectatic abscess caused by the impaction of an O'Dwyer's tube, which had slipped down the larynx during a paroxysm of cough and lodged in the left bronchus three-fourths of an inch below the bifurcation of the trachea.

Sir Dyce Duckworth,[74] in a clinical lecture on the subject recorded in the *Clinical Journal*, 1892, went over the pathological anatomy, symptoms, physical signs, and treatment of bronchiectasis. He did not advise surgical interference.

In June, 1893,[75] Grainger Stewart recorded a typical case of the disease treated with marked success by the intra-tracheal injection of menthol, guaiacol, and olive oil, and strongly recommended that this treatment should be tried in other cases.

Ewart,[76] commenting on a case which he records, lays great stress on some essential particulars in which a bronchiectatic patient differs from a phthisical—viz., the absence of emaciation, the absence of anæmia, and the presence of a more or less *venous* condition. This latter, as well as the clubbing of the fingers, so often found in patients suffering from this disease, he thinks a result of the resistance in the pulmonary circulation and the destruction of the smaller bronchial vessels which form a connecting link between the pulmonary and systemic circulations. The cause of the disease, he thinks, is always some obstruction, and that obstruction may be situated *in fronte* as in bronchial stenosis, or *in tergo*, as in bronchial catarrh. Bronchiectasis and emphysema are in his opinion complementary.

Sir Andrew Clark[77] devoted some attention to bronchiectasis as it occurs in association with fibroid disease of the lungs, to which he thought it is secondary. He mentioned that in fibrosis, dilatations are so common that before the time of Corrigan it was believed that the dilatation of the tubes was primary, the fibrosis secondary.

Morbid Anatomy.

The most common seats of bronchiectases are the lower lobes of both lungs and the middle lobe of the right. It occasionally occurs

toward the apices. Usually it affects many bronchia in both lungs. Examples of the disease present a great variety of morbid appearances, and in order to make this subject clear, it is necessary to describe the conditions:

I. Of the bronchia themselves;
II. Of their contents;
III. Of the surrounding pulmonary tissue;
IV. Of the pleura;
V. Of the thoracic walls.

I. Changes in the Bronchial Walls.

A. General.—(*a*) In some instances we find a dilatation in a single tube and at a single point. This is generally equally distributed in the whole circumference of the bronchial wall, but sometimes is more marked at or even confined to one side. Such dilatations are most commonly associated with stricture of the tube, and may be found on either its distal or its proximal side—sometimes on both.

(*b*) We find occasionally that a large number of the minuter bronchia are widened out, not into large spaces, but yet presenting throughout a considerable extent of their length a dilatation, so that the tubes, instead of becoming smaller as they advance, remain of the same size nearly to the infundibula, showing in that position a calibre as great as that which they had shown at the second or third division. This form is met with in association with whooping-cough, and with capillary bronchitis, and sometimes apart from these conditions.

(*c*) We meet with dilatations, sometimes affecting one, sometimes a number of tubes, where the contour is somewhat irregular; this condition is unimportant as compared with the changes with which it is associated; it occurs in cirrhosis of the lung, and sometimes in other chronic forms of phthisis.

(*d*) We meet with cases which may be termed spurious bronchiectasis, where cavities communicating with the bronchia may be and have been mistaken for true dilatations; these occur in connection with phthisis pulmonalis.

(*e*) The most important of all the varieties is that in which as a rule many bronchial tubes are dilated, the tendency manifesting itself about the second or third division and continuing throughout the course of the tubes in what may be termed a cylindro-ampullary manner—the whole cavity being enlarged, but certain portions more markedly so; occasionally this enlargement is distinctly bead-like or moniliform, more commonly not so. But even when the disease affects many tubes, certain individual ones are more particularly involved; and in these the chief pathological and clinical phenomena centre.

Not unfrequently neighboring dilatations communicate with one another, and there are ridges traceable in the walls here and there, and even bridges which are described in detail further on.

B. Changes in the Mucous Membrane.—In many cases the mucous membrane appears to the naked eye and microscopically absolutely normal, its ciliated epithelium and other layers presenting precisely the conditions of health. But more commonly alterations are observed, and among these we notice:

1. Thinning and atrophy. To the naked eye the mucous membrane appears thinner, more delicate, and more glistening than usual, and on microscopic examination the epithelium is found of smaller size, the cilia are less distinct or may be wanting, the surface presenting a pavement-like appearance.

2. The membrane often shows inflammatory thickening; even to the naked eye it may appear villous and congested, and on microscopic examination its vessels are overfilled; there is a cloudy and altered condition of the cellular elements, and sometimes an irregular roughening of the surface, a result of the inflammatory action.

3. Occasionally the inflammatory products form a layer of false membrane, which may cover the surface as a thin film, or may block the lumen of the tube. It is not very uncommon to find evidence of such inflammation in bronchioles which have not become affected with dilatation, these being plugged with a fibrinous material as in an ordinary case of plastic bronchitis.

4. Ulceration of a superficial kind is to be found here and there in some of the dilatations usually associated with the inflammatory thickenings just described.

5. In rare instances gangrenous destruction of the mucous membrane exists—this not generally widespread, but confined to limited areas.

C. Changes in Muscular, Elastic, and Cartilaginous Structures.—1. Even in the slighter dilatations in which the mucous membrane is quite normal, there is distinct wasting of the muscular and elastic coats. On microscopic examination it often happens that the histological elements can scarcely be made out. The cartilages become smaller and thinner as the other tissues diminish, and they are said sometimes entirely to disappear.

2. In other instances these layers are found to have undergone a thickening. They present a condition which may be termed one of spurious hypertrophy. The inflammatory process has involved the walls, and in these it sometimes happens that there is evidence of special irritation of the cartilaginous plates. Andral and Biermer spoke of outgrowths from them and proliferation of the cartilage cells

is often mentioned. We have seen this only in cases where the mucous membrane also exhibited corresponding alteration.

3. A gangrenous change sometimes occurs in these deeper elements of the walls; and we have seen the thickened fibrous layer retaining its continuity when gangrene had set in in the surrounding tissue, so that the partially broken-down tubes could be traced among the tissue débris.

D. Formation of Communications Between Neighboring Bronchiectases.—Laennec, Cruveilhier, and others have described this condition, and although its existence was denied by Hasse and is not referred to by many writers on the subject, we have met with it so often as to leave us in no doubt about its occurrence. The communications are sometimes small, such as would admit a crow-quill; at other times they are half an inch or even more in length, and up to half an inch in width. The process of their formation is peculiarly interesting. Neighboring bronchiectases come into contact with one another back to back, they become adherent, and a process of absorption leads to the formation of an opening, at first of a minute size but gradually enlarging. We have never seen a roughened or ulcerated edge, but always a smooth, shining, and continuous one; the process of atrophy leads to the absorption first of the deeper tissues of the wall, so that only basement membrane with its epithelial covering is for the time left, and, as fenestration takes place, the epithelium at the two sides becomes continuous and the edge gradually recedes, the opening becoming progressively larger.

E. The Formation of Ridges on the Walls and Bridges Within the Lumen of the Tube.—It almost never happens that the dilatation in a bronchus proceeds equally at all points in the circumference, but not infrequently portions fail to be dilated, and as the neighboring structures recede these unchanged portions necessarily project from the adjoining walls. The epithelial covering remains quite continuous, and in such ridges the wasting proper to the deeper tissues does not occur; but as the process goes on, the ridge becomes so prominent that beneath it, and connecting it with the wall, there is left only a double layer of mucous membrane. This makes a mesentery-like attachment to the wall; but it gradually thins away and fenestrations appear in it—no ulceration, but simply a gradual absorption with smooth and even edges. And this fenestration enlarges until the prominent unatrophied part is separated from the wall, except at its two ends, and forms a bridge within the lumen of the tube. This condition, which was first described by one of us in 1867, has been recognized by Auld and others; but Auld has arrived at a different conclusion regarding its mode of origin. He believes that the bridges

are formed by the fusion of the apices of papillary outgrowths from the mucous membrane, and he claims to have observed in one case various stages of this process. Our own observations and the analogy of pathological processes lead us decidedly to prefer the above explanation to that of Dr. Auld.

II. Condition of the Contents of the Tubes.

The dilatations usually contain much mucus, but in different cases and in different parts of the same lung it may be found in very different conditions.

1. There is in some cases a copious yellow mucus quite free from fœtor and presenting the characters of the natural secretion both to the naked eye and on microscopic examination. On agitation with water, casts of smaller bronchi containing much epithelium may be separated, the epithelium presenting a natural or only slightly granular appearance.

2. In other cases we find an inspissated mucus of a gray yellow or rather opaque color usually free from any disagreeable odor and frequently partially adhering to the walls. On microscopic examination some altered cells may be recognized in this material, but it is mostly composed of granular and fatty matter.

3. In other cases the mucus is decomposed and very fetid, contains fatty granules, altered epithelium, and fine needle-shaped crystals of margarin; casts of tubes are also present, and these often, as was first shown by Dr. Arthur Gamgee, assume a purplish color on the addition of iodine.

4. Blood is sometimes mingled with the mucus, in addition to the different elements already referred to.

5. We find occasionally that elements of pulmonary tissue may be distinguished among these other materials.

6. The contents sometimes become inspissated, hard, and even calcareous.

It is obvious that most of these changes result from alteration of the natural bronchial secretion. It has been experimentally proved that if the mucus be kept in a bottle at the temperature of the body, it undergoes decomposition and develops a fœtor corresponding to that of bronchiectasis. The cases in which blood, casts, or fragments of pulmonary tissue are present are those in which an extension of the malady has taken place so as to involve other structures beyond.

As to the chemistry of the sputum, we have in the mean time little information—the discussion has turned mainly upon the question of the cause of the fœtor. Professor Laycock concluded from some

experiments that the fetor must be due to butyric acid, and he stated that Dr. Gregory detected the odor of methylamine in some of the products of the sputa. Bamberger concludes that the characteristic smell of the sputa in bronchiectasis appears to depend on a variety of odorous matters, among which are the members of the series of acids of the type $C_nH_nO_4$ particularly butyric, and also formic acid, ammonia and sulphuretted hydrogen, all of which may proceed from the decomposition of organic substances. The taste of the sputum in the patient's mouth is said by Williams to be sweet.

III. Condition of the Surrounding Pulmonary Tissues.

In a considerable number of cases the pulmonary tissue surrounding dilated bronchi is soft, spongy, and crepitant, presenting to the naked eye as well as on microscopic examination normal appearances.

In other instances it is found collapsed or atrophied; the tissue is not spongy and does not crepitate, but it is not indurated, and on microscopic examination no excess of fibrous tissue is to be detected.

In not a few cases the tissue is consolidated, and of this condition we may recognize several varieties:

(*a*) A condition of chronic fibroid change, spoken of by some as cirrhosis and by others as fibroid degeneration of lung;

(*b*) A similar induration but with distinct tubercular character, generally a chronic form of tubercular phthisis;

(*c*) An inflammatory consolidation of the pulmonary tissue, generally of a catarrhal nature, this extending to a varying depth into the tissue around the dilatations.

Occasionally one finds that suppuration has taken place in the pulmonary tissue surrounding a dilated bronchus; and the walls with their spurious hypertrophy may sometimes be traced extending through the abscess.

Not very rarely the walls of the bronchi and the surrounding lung tissue are gangrenous, an offensive greenish débris representing the gangrenous destruction of pulmonary tissue.

IV. Condition of the Pleura.

The two layers of the pleura are in many cases adherent and sometimes are thickened to an extraordinary extent. But in a considerable proportion of cases there is no such thickening, and not unfrequently the pleura is normal.

V. The Form of the Thorax.

There is no constant alteration in this respect; but it may be found rounded, as in some cases of emphysema, and sometimes shrunken throughout more or less extended areas. We have known it marked by a peculiar roundness of the shoulders.

Pathogenesis.

Since the time when Laennec, in bringing to the knowledge of the profession the existence of dilatations of the bronchi, attempted to find their explanation in stagnation of secretion and mechanical pressure on their walls, the theories put forward to explain the various conditions which have come to be included in the term "bronchiectasis" have been very numerous. Their number and variety may be gathered from the preceding historical sketch. But although most of the writers have expressed more or less different views as to either the pathological or the mechanical factors concerned in the production of these conditions we may broadly group together the theories as follows:

1. Mechanical Pressure of Stagnating Secretion.

This was the view suggested by Laennec, and in which he was followed by Andral, Cruveilhier, and others. They held that accumulation of mucus distended and dilated the bronchi, the process being aided, according to the later writers, by cough.

Several difficulties stand in the way of the acceptance of this view:

1st. In many bronchiectases no special accumulation of mucus is found after death; and there is no indication of its existence at an earlier stage of the process;

2d. Even if this were the case it is mechanically impossible that dilatation should result from it, considering the consistence of the secretion and the free outlet which exists in two directions.

2. Concentrated Air Pressure.

This view, originally enunciated by Reynaud who considered the dilatations compensatory to atrophy or collapse of neighboring parts, the pressure of the inspired air acting with greater force on the diminished respiratory surface, was adopted and elaborated by the elder Williams. He pointed out that if the equality of pressure be disturbed by more forcible pressure of the air on individual parts of the respiratory tract or on all parts, dilatations of the parts so pressed

upon must occur, a gradual impairment taking place in respect to their elasticity and contractility.

The difficultes which forbid the acceptance of this theory are:

1st. That the only conditions under which increased air pressure exists are those associated with violent coughing, when powerful expiratory effort is made, the glottis being at the beginning of the act closed; now, on the one hand, bronchiectasis arises without preexisting cough; and on the other, cough is extremely common, while bronchiectasis is rare;

2d. That even where such a cause does operate there must be an unusual debility of the bronchial walls; for they are naturally less yielding than the pulmonary tissue, and emphysema rather than bronchiectasis would result.

3. Extra-Bronchial Traction.

This is the view of Corrigan, Barth, and more recently of Hamilton. These observers suppose the dilatations to result from traction upon the bronchial walls by contracting cirrhosed lung tissue, aided by, according to Barth and Hamilton, pleuritic adhesions to the chest wall.

The objections to the exclusive adoption of this theory are:

1st. That bronchiectasis often arises where there is no cirrhosis of the lung, and no adhesions of the pleura;

2d. That in cases of bronchiectasis with pulmonary consolidation it is sometimes possible to demonstrate that the dilatation preceded in order of time the condensation of the lung;

3d. That the process of traction, while it might account for dilatations, could never explain the opening of neighboring dilatations into one another, nor the formation of bridges in dilated portions, both of which are often seen in the midst of consolidated pulmonary tissue;

4th. That cirrhosis is frequently unaccompanied by bronchiectasis; according to Bastian there is no dilatation in twenty per cent. of such cases.

4. Inflammation of the Bronchial Wall.

Inflammatory changes in the bronchial walls have been considered of more or less importance in the causation of the disease from the time of Laennec downwards; but it was Dr. Stokes, of Dublin, who first clearly enunciated the theory that as a result of inflammation the bronchial walls lose their elasticity, contractility, and ciliary movement, and consequently dilate, and so accumulation of secre-

tion occurs. This view has been adopted by several writers and most recently by Wilson Fox.

The chief objections to it are:

1st. That if bronchitis be capable of inducing bronchiectasis, the condition ought to be much more common than it is;

2d. That it seems certain that in some cases bronchiectasis has developed without previously existing bronchitis;

3d. That the walls of dilatations not unfrequently are found simply atrophied and without any token of inflammation.

5. *Nervous Influence.*

Lebert, though generally concurring in the views of Stokes. thought the loss of power dependent upon nervous causes rather than upon inflammation of the bronchi.

The objections to this are:

1st. That it is purely hypothetical and gratuitous;

2d. That in such analogous processes as aneurysm, varicosity of veins, and staphyloma posticum there is no reason to think that the nervous system plays a part, but rather that local nutritive changes are the main causes.

6. *Primary Atrophy of the Bronchial Wall.*

This view was definitely presented in a paper published in 1867 by one of the present writers. It was founded upon a series of observations, pathological and clinical, for the most part corresponding to the views expressed in this article, the writer's further observations having confirmed the impression then formed. It is of course to be understood that this view is offered as explanation of one form of bronchiectasis only—but that the chief and most important form—while the existence of other varieties, which he would prefer to call *secondary* bronchiectases, is fully admitted. The process, then, is to be regarded as analogous to the direct atrophic form of emphysema, and not improbably to result from a constitutional, possibly an hereditary defect.

The same view has to a large extent and apparenly independently been arrived at by Leroy as described in a paper already referred to.

The view, then, which according to present information appears most satisfactory is that bronchiectases may be classified in the following way, viz.:

A. Primary, and

B. Secondary.

A. *Primary bronchiectasis* is a disease of the bronchi, the essen-

cial element of which is *atrophy* of the bronchial walls; the cause of such atrophy is not yet understood, but is probably connected with constitutional peculiarities.

The walls of the bronchi being so thinned and weakened yield readily to the *ordinary* air pressure; or it may be that deep and sudden inspirations, or violent muscular exercise, or the sudden expiratory efforts made while the glottis is closed in the act of coughing, aid the process of dilatation.

The enfeebled and dilated condition of the bronchial tubes favors the accumulation of mucus secreted by their mucous membrane.

The mucus accumulating and undergoing decomposition in the dilatations irritates the mucous membrane, leads to inflammation and the formation of villous processes, to the formation of increased connective tissue, to irritation of the cartilages, and frequently to consolidation of the surrounding lung tissue, and pleuritic adhesions; sometimes also to abscess or to limited gangrene.

The process then would appear to correspond in its early stages to what is met with in other parts of the body, as, for example, in true aneurysm, where from weakening of the middle coat of the artery the pressure of the blood causes the artery to expand, and that to a constantly increasing extent. It also resembles the process by which staphyloma of the sclerotic is formed, in which the membrane, being weakened at certain points, is unable to withstand the pressure of the fluids of the globe, and consequently a bulging ensues.

B. Secondary bronchiectasis is a dilatation of the bronchi occurring in the course of various morbid conditions of the surrounding pulmonary tissues, or caused by mechanical obstruction to the free passage of the air in inspiration or in expiration.

Secondary bronchiectasis may be

1. General, or
2. Local.

1. General, as in the finger-like dilatations occasionally found after death from whooping-cough and capillary bronchitis.

2. Local, as in the dilatations found (*a*) in cirrhosed lungs; or (*b*) in relation to a stenosis of a bronchus, whether the stenosis be due to stricture or to compression from an aneurysm, tumor, or enlarged glands; or (*c*) in relation to an impacted foreign body.

It is to these secondary varieties of bronchiectasis that the explanations accepted by most writers must be held to apply. It is indeed difficult to assign to each factor suggested its proper influence in the production of the dilatations; for when we consider the combination of morbid appearances which is often found in the post-mortem room in a single case, we are led to conclude that the mech-

anism of its production is also a complex one. But if we confine our attention to the external forces to which the bronchial tubes may be subjected, and to their respective parts in the mechanism of dilatation, we cannot arrive at a true understanding of the pathology of these affections. The bronchi, which are contractile and resilient tubes, are strictly analogous to the arteries and veins. And just as an artery does not dilate to become an aneurysm, or a vein to become permanently varicose, so long as it is healthy, that is, so long as the integrity and resiliency of the vessel are maintained, neither will a bronchus with its integrity and resiliency intact yield permanently to such forces as can be brought to bear upon it. And what is true of the primary variety of bronchiectasis is therefore in a measure true of these secondary dilatations. The source of the weakness, and therefore the starting-point of the dilatation, is some form of inflammation, either acute or chronic, and affecting any of the coats of the wall. Inflammation weakens the bronchus in two ways, whether thickening or thinning be the obvious result: namely, by destruction of some of the elements of the wall—thus impairing its *integrity;* or by the organization of inflammatory products—thus impairing its *resiliency.* From the analogy of the pathology of dilatation in other tubes we know that any impairment of the middle muscular, an elastic, contractile, and most resilient coat, is a source of much greater weakness than impairment of either the inner mucous or the outer fibrous coat; and this fact doubtless explains the rarity of bronchiectasis compared with the frequency of bronchial inflammations, which mainly involve the mucous membrane. There must therefore be some weakness of the bronchial wall to permit of dilatation occurring under the influence of such extraneous forces as the traction of contracting fibrous tissue, or the pressure of air compressed by the expiratory efforts of cough or in forced inspiration. How much influence to assign to each of these in any given case is, as we have already remarked, difficult to determine. We may, however, say that it is difficult to imagine the possibility of any increase of inspiratory pressure such as shall surpass the atmospheric pressure; whereas Mendelssohn has computed that in the act of coughing, the vocal cords being closed, the air is compressed, and the expiratory pressure may be increased by one-seventh atmosphere more.

For the physician, however, speculation as to these matters is of less importance than the recognition of the fact on which we desire to insist, that permanent dilatation will not take place in a healthy bronchus, but in a bronchus weakened by inflammatory changes which it is possible sometimes to prevent and often to modify.

When dilatation has taken place, the secondary changes in the

contents, in the walls, and in the surrounding lung tissues, are of the same nature as those associated with primary bronchiectasis.

Clinical History.

It follows of necessity from the anatomical facts which have been described that bronchiectasis may be associated with a great variety of clinical features. In what we have termed the *secondary* forms of the disease—those following upon whooping-cough, capillary bronchitis, cirrhosis of the lungs, and such like changes—the features of these diseases generally overshadow those proper to the bronchial dilatations, and it is only when retention and decomposition of sputum have taken place that the diagnosis can be established.

In the case of what we have termed primary bronchiectasis, the symptoms are often in the early stages indistinguishable; but as the disease advances, they become unmistakably distinct.

We shall follow out briefly the history of typical examples of the secondary forms, and then shall describe in detail the features of the primary variety.

1. A child two or three years old has had measles in infancy; and the attendant bronchial catarrh has not cleared away when whooping-cough supervenes, reducing the child's strength, and leaving it with a wheezy chest and a chronic cough. Frequent attacks of bronchitis in the larger or in the capillary tubes further exhaust the child; it becomes emaciated; there is more or less cyanosis; the fingers and toes become clubbed; and there is so much cough that the child is probably treated for phthisis. The breath and expectoration may become more or less fetid; the cough is spasmodic and choking; but rarely does the child expectorate in the way which is so characteristic in the adult, for if the sputum is brought up it is very generally swallowed unless when an act of vomiting attends it. The chest becomes deformed, with recession below the nipples, perhaps unduly resonant all over, perhaps with a consolidated area at one or both bases. The breathing is tubular and blowing and there are numerous consonating râles mixed with the coarser sounds of bronchitis. The child dies from exhaustion or an intercurrent attack of pneumonia. On post-mortem examination, one or both lungs are found on section to be riddled with cavities which turn out to be not phthisical vomicæ, but dilated bronchial tubes; the intervening lung parenchyma being in various stages of inflammation and condensation.

2. A patient has had fibroid phthisis of one lung for many years, manifested by the physical signs dyspnœa, chronic cough, occasional

attacks of hæmoptysis, clubbing of the fingers, etc. He has occasional exacerbations of pulmonary symptoms, and during one of these his expectoration gradually increases and alters in character, so that at last it amounts to half a pint or one pint daily, and presents a peculiar greenish-yellow muco-purulent appearance; and it comes up in mouthfuls. From that time all his symptoms become worse, and this expectoration becomes occasionally fetid. If this fœtor be once established he has serious febrile attacks. The patient may under treatment improve to such an extent as to be able to go about his work, but he is always liable to recurrence of attacks from slight causes, and these attacks are more severe and attended by a return of increased and offensive expectoration. He ultimately succumbs to pneumonia, gangrene, or septicæmia. On post-mortem examination one lung is found to show great compensatory enlargement; the other is reduced in size and much condensed, and throughout it, in addition to the ordinary evidences of fibroid phthisis and the immediate intercurrent complication, are found pouches of dilated bronchi, the walls of which are probably thickened and fibrous.

3. A very typical case of bronchiectasis due to the impaction of a foreign body in a bronchus, is related by Dr. G. R. Sutherland. A youth, aged 17½, was being treated with an O'Dwyer's tube for laryngeal dyspnœa, the result of an injury to the vagus in the neck. During a fit of coughing the attaching cord broke, and the tube slipped down the larynx and trachea and became impacted in the left bronchus. About two months afterwards he began to suffer from a very copious purulent expectoration, which, with a bad cough, was the only symptom of active mischief. A month later the expectoration became intermittent and very fetid; and loud blowing breathing became apparent at the inferior angle of the left scapula, with metallic râles and whispering pectoriloquy. The patient died after an operation for draining the cavity which had become established. The right lung was found hypertrophied and emphysematous. In the left there was below the obstruction one large cavity, the walls of which were formed by the dilated bronchus, the greater part of the lower lobe having been destroyed. The bronchi around were also dilated and surrounded by soft and easily broken down lung tissue.

Primary Bronchiectasis.

In many, if not in all cases, the affection of the bronchi comes on insidiously, no symptoms appearing to attract the attention of the patient or his friends, perhaps for long after the dilatations have been formed. But gradually or suddenly inflammation of the bronchial

mucous membrane is lighted up, and symptoms of bronchitis become well marked. Those of bronchiectasis, though distinct when looked for, are not such as to force themselves on the attention until decomposition of the secretion has taken place; the breath and sputum then become fetid, and the general health obviously suffers. The patient's history is now one of intermittent cough, with fetid sputum and breath; occasional feverish attacks and exacerbations of cold, during which all his symptoms become very much worse. He also becomes liable to more severe febrile disturbances due to a septicæmia from the absorption of the products of decomposition, which attacks may be recovered from under treatment, or may prove fatal; or the decomposition of the secretion may be followed by inflammation and consolidation of the lung—a septic pneumonia; sometimes also by ulceration, abscess, or gangrene. Following upon these, we sometimes have a fatal result from perforation of the pleura, leading to empyema and pneumothorax. Sometimes death results from exhaustion induced by the constant discharge of sputum.

A typical case of primary bronchiectasis with features fully developed will present the following clinical phenomena:

The patient, who is usually a male, middle-aged, the disease being rare in women, comes complaining of cough and fœtor of breath, perhaps of feverishness and general malaise.

He may not be able to give any facts to show an hereditary tendency; the disease is usually stated to be non-hereditary, and not constitutional in its origin. But from a consideration of its nature, and of the facts of certain cases, it appears probable that it may ultimately prove to be both. We have known of two cases occurring in members of the same family.

As a rule, except in relation to an acute febrile attack, there is not much emaciation. On the contrary, the patient is well nourished; he has rounded shoulders and a tendency to cyanosis. The fingers and toes are often clubbed; the complexion is dirty and the appearance of the skin is oily.

The temperature except during acute attacks is normal or subnormal. The breath is offensive, and there is much cough with profuse fetid expectoration, so fetid often as to be oppressive and unbearable to the patient's companions.

The alimentary system is often badly deranged; the tongue is foul; the appetite and digestion are impaired.

The hæmatopoietic system shows no special change.

The circulatory system often shows evidence of some obstruction to the pulmonary circulation. There is a tendency to enlargement of the right side of the heart, and to cyanosis and clubbing of the

fingers. The pulse is generally rapid, even independently of the occurrence of inflammatory complications.

The chief clinical features centre in the respiratory system. The breath is often peculiarly foul; the fœtor somewhat different from that of the sputum and is often intensely disagreeable to the patient himself. It is difficult to indicate a smell closely corresponding to it; but it is certainly quite distinct from that of gangrene. This symptom is not present in all cases, nor is it present in the earlier stages of any case. It is only as the disease advances, and when decomposition of the bronchial secretion has taken place, that it occurs.

The *sputum* is usually copious—fifteen to thirty ounces daily—of a greenish-yellow color and nummular, rarely frothy on the surface; sometimes gelatinous and with a tendency to adhere to the walls of the vessel. Sometimes it sinks, sometimes floats in water; and when a quantity of it is allowed to stand in a glass jar it separates into three layers or strata—a more mucous and fatty layer which floats on the surface; a more purulent and granular matter which sinks to the bottom, and a greenish watery fluid in which the other two are embedded, but which forms an intermediate layer.

In a majority of cases—at least of cases in which the disease is recognized—the sputum is very fetid. The fœtor resembles that of the breath but in a more concentrated form. It is quite distinct from the odor proper to gangrene. On agitation of the recent sputum with water, more opaque filaments of varying thickness separate themselves and sink to the bottom. These are casts of bronchial tubes.

On microscopic examination the sputum shows large numbers of pus corpuscles, oil globules, fatty crystals, and occasionally, when ulceration is going on, fragments of pulmonary elastic tissue. Cholesterin crystals also occur. It may be found to contain sarcinæ, the mycosis aspergillina of Virchow, and numerous other micro-organisms.

Hæmoptysis.—Blood is expectorated along with the sputum, sometimes in small quantities merely streaking the expectoration which otherwise presents the characters above described; but sometimes it is brought up in large quantities much as in phthisis. In such cases the sputum may not be at all abundant; it is only when the hemorrhage occurs that barely any discharge takes place. Barth met with two cases in which the hæmoptysis proved fatal, and we have met with similar cases in the post-mortem theatre and in practice.

Cough is frequent, and occurs in paroxysms, the fits of coughing being peculiarly prolonged and at first resultless, but ultimately attended by the discharge of large quantities of the green-yellow sputum. The cough is as a rule moist and usually quite painless.

Respiration is generally little disturbed while the patient is at rest, but exertion speedily induces dyspnœa with laborious breathing. In advanced cases, where the dilatations have destroyed or replaced large portions of pulmonary tissue, dyspnœa is a constant symptom.

The form of the chest is not necessarily altered, although the shoulders are often rounded. At times, and depending more on secondary affections than on the primary lesion, there may be evident retraction in part, and diminished mobility.

Palpation.—Vocal fremitus is as a rule increased over the bronchiectatic areas; but the palpation phenomena depend more on the condition of the pulmonary tissue than on the bronchiectases themselves.

Percussion.—In the earlier stages the percussion note is little altered, and even in advanced conditions there may not be much change. There is in advanced stages dulness on percussion over the parts of the lung which have become consolidated, sometimes relieved by an unusually clear note if a dilatation be situated near the surface and empty. While the dilatations may occur in any part of the chest, they are met with most frequently posteriorly, at and below the roots of the lungs. And so we find that the most characteristic percussion phenomena in a well-marked case are a high-pitched percussion note, sometimes tympanitic, sometimes even cracked-pot in these situations, with a varying but usually small zone of dulness round about.

Auscultation.—The auscultatory phenomena are very important elements in the diagnosis of the condition.

1. There are the ordinary signs of bronchitis in most cases.

2. At various points, but most frequently about the roots of the lungs and toward the bases, the breath sounds are cavernous. The characters of these have been variously described by different writers as tubular, amphoric, nasal, croaking, jarring—characters which evidently depend on the condition of the cavities as to shape, size, and amount of contents.

3. Accompanying these breath sounds are a variety of râles: coarse, fine, bubbling, and even gurgling. A noteworthy feature of the accompaniments is their variability; they are at one time unmistakably distinct; at another time, when the cavity or cavities have been emptied after a fit of coughing, they are indistinct or absent.

4. Over these same areas the vocal resonance is bronchophonic or more frequently pectoriloquous, and often peculiarly harsh and jarring.

The physical phenomena are briefly then unmistakable signs of cavity formation, in unusual situations, and with or without evidence of any considerable consolidation of lung tissue.

In the integumentary system we notice the dirtiness of the complexion and the oiliness of the skin.

The urinary, nervous, and locomotor systems are not necessarily deranged.

Complications and Sequelæ.

1. As a result of the decomposition of secretion retained in the dilatations, and the absorption of the products of decomposition, bronchiectatic patients frequently exhibit general febrile disturbances of the nature of a sapræmia or a septicæmia.

2. Metastatic abscesses, especially cerebral, and septic peritonitis have also been observed (Dittrich).

3. In consequence of either the irritation of the decomposing secretion, or septic infection, various local complications may arise:

(*a*) Pneumonia;

(*b*) Abscess of the lung;

(*c*) Gangrene of the lung;

(*d*) Pleurisy and empyema, and pneumothorax from ulceration.

In such cases the symptoms and physical signs proper to the complication are superadded to those peculiar to bronchiectasis.

4. Pulmonary fibrosis is, as we know from clinical experience, a sequel of bronchiectatic dilatations.

5. Calcareous changes in the walls and contents are sometimes observed, but are not of clinical importance.

Diagnosis.

The conditions which are apt to be confounded with bronchiectasis are:

(*a*) Fetid bronchitis.

(*b*) Phthisis pulmonalis including fibroid phthisis.

(*c*) Gangrene of the lung.

(*d*) Carcinomatous ulceration of the lung.

(*e*) Syphilitic ulceration of the lung.

(*f*) Limited empyema communicating with a bronchus.

(*a*) *Fetid Bronchitis.*—This is a rare condition in which either permanently, temporarily, or occasionally, fœtor of breath and of sputum becomes added to the ordinary features of bronchitis, subacute or chronic. The distinction is not difficult in the merely temporary or the recurrent cases because there is no physical sign indicative of the presence of a cavity, and because the fœtor either rapidly disappears or comes on occasionally. It may be found on close examination that the fœtor in such cases is due to primary change in the sputum, and sometimes to constitutional conditions

attended by faulty chemical processes in the liver, the intestinal canal, or elsewhere. So it sometimes happens that the inhalation or intratracheal injection of antiseptics relieves the condition, and that sometimes a few doses of aperient or cholagogue medicine suffice to remove the symptom; and then the bronchitis shows itself in a simple condition amenable to ordinary treatment. Under such circumstances the physician is rarely at a loss for diagnosis. But when the fœtor is constant, the sputum rather copious and presenting a greenish-yellow color and somewhat oily consistence, diagnosis is far more difficult, for one has then to depend mainly upon the physical signs of cavities, and it is obvious that these might be absent in any case in which the cavities with the bronchi affected were somewhat deeply seated. In such cases it is impossible to decide with positive certainty, and an alternative diagnosis is generally to be preferred to a positive one.

(*b*) The cases of *phthisis* which are most apt to be confounded with bronchiectasis are those in which fœtor of breath and sputum is developed in consequence of decomposition of the secretion.

The differential diagnosis turns—

1. Upon the physical signs of the chest;
2. Upon the examination of the sputum.

The physical signs which are of value are those which indicate the seat of the cavity formation. Whereas in phthisis the vomicæ are most pronounced toward the apices, in bronchiectasis they are seated near the base, or at all events toward the lower parts of the lung. The middle lobe of the right and the lower lobes of both lungs are much more frequently involved than are the bronchial branches in the upper lobes. Another point of some value is the fact that we sometimes get in bronchiectasis distinct cavernous breathing without evidence of pulmonary consolidation, which of course we scarcely ever meet with in phthisis.

The examination of the sputum brings out in the case of phthisis, not only fragments of pulmonary tissue, but the tubercle bacillus as well; and both of these are, in cases liable to be confounded with bronchiectasis, as a rule extremely numerous.

With regard to the distinction between primary bronchiectasis and the secondary dilatations which occur in the course of fibroid phthisis, the history of the case will help us. The physical signs also proper to fibroid phthisis, and the occurrence of tubercle bacilli if they be present in the particular case, leave us in little doubt.

Besides these we derive considerable assistance from a study of the general condition of the patient as regards emaciation, of the temperature curve, and of the character of the cough.

(*c*) When *gangrene of the lungs* is attended with fœtor of breath, it may be confounded with bronchiectasis, but the distinction is not as a rule difficult. Gangrene usually sets in as the result of an acute process such as pneumonia or embolism. It is not attended by the peculiar cough or characteristic sputum of bronchiectasis. The fœtor of the breath and expectoration is quite distinct from that proper to bronchiectasis, and the physical signs of vomicæ are almost never to be made out; while, on the other hand, the depression of vital power is far more marked and more speedily developed in gangrene than it ever is in bronchiectasis.

But it must be remembered that gangrene occasionally becomes superadded to the bronchial dilatation; and we have known the occurrence of this to mark itself by sharp hæmoptysis, and by a sudden change in the odor and the appearance of the expectoration, so that a case which had at one time shown the characteristic symptoms and signs of bronchiectasis came within a few days to show those proper to gangrene. As this combination is almost necessarily fatal, we frequently obtain a speedy confirmation of our diagnosis.

(*d*) *Carcinoma of the Lung.*—In a small proportion of cases of pulmonary carcinoma fœtor of breath occurs as a complication; and in such cases it is conceivable that confusion of diagnosis might arise. But the history of carcinoma, its physical signs, and the general condition of the patient suffice to mark it off definitely from the other condition.

(*e*) *Syphilitic affections* of the air passages may be confounded with bronchiectasis when they are attended by marked fœtor. But the syphilitic constitution and the local manifestations of syphilitic disease elsewhere, taken along with the absence of the physical signs proper to bronchiectasis, generally suffice to mark these conditions off with sufficient distinctness.

(*f*) *Limited Empyema.*—If we conceive of an empyema between the lobes of the lung, and discharging through the pulmonary tissue and through a bronchus, it is obvious that we get a condition very closely resembling bronchiectasis. The history of such cases is often so obscure as to afford little help in diagnosis. But the position of the cavity, the character of the physical signs, and the other features of the sputum will generally suffice for differential diagnosis. We are most inclined to insist on the character of the sputum; if it be derived from a pneumo-empyema cavity, it is more purely purulent, and has not the oily appearance, nor the admixture of mucus and of fibrinous casts of the minute tubes; and we have never seen fatty crystals in such cases.

Prognosis.

It is probable that some cases of bronchial dilatation due to whooping-cough or capillary bronchitis may gradually subside when these maladies have passed away. But in a great majority of cases the disease tends to a fatal result, although now and then recovery is said to occur. Gintrac relates that recovery has been observed to come about in two ways, each of them sufficiently remarkable: 1st From the cretification of the contents, and the conversion of the walls into a sort of fibrous capsule; 2d. From penetration of the pleura and thoracic parietes, and subsequent discharge of the contents.

While recoveries by such means must be exceedingly rare (and we have never met with anything corresponding to them), we have ourselves seen, and there are several other cases on record, recoveries apparently permanent after surgical interference. And furthermore, by suitable means we sometimes succeed in so checking the decomposition and the amount of secretion that the disease may be compatible with fairly good health for many years. But considering the views above expressed as to the morbid anatomy and pathology of the process, it is obvious that we cannot be sanguine as to its disappearance.

Treatment.

The treatment of bronchiectasis is a matter in which considerable progress has been made in recent years; and several methods which have been of marked service in the few cases in which they have been tried are deserving of more extensive application and study.

A consideration of the pathology and clinical features of the disease and of its many complications makes it evident that our efforts have to be employed in several directions.

Prophylaxis.

It is to be feared that little can be accomplished in the way of *prophylaxis* of the disease; for it is not in our power at present to prevent the occurrence of the atrophy which is the essential element in the primary form of bronchiectasis; and a recognition of the importance of bronchial inflammations—of such diseases as whooping-cough and capillary bronchitis—in the production of the secondary varieties, can hardly stimulate us to bestow upon them more attention and care than these conditions demand on their own account.

Treatment of the Results of the Disease.

We are called upon therefore most frequently to treat the fully established disease and its results; and our efforts have to be directed toward preventing, as far as possible, the extension of the cavities and the development of those changes in their contents on which the main clinical features depend.

It is important to prevent the occurrence of fresh attacks of bronchial catarrh, and to keep down cough as far as that is warrantable. Where possible, therefore, climatic treatment has to be kept in view.

Excessive secretion may be relieved by the administration of astringents and balsamic substances, either by the stomach or by inhalation; and it often yields to counter-irritation, especially by blisters.

Various methods, the aim of which has been to prevent decomposition of the sputum, have been tried from time to time:

(*a*) The administration of various antiseptics by the stomach, such as turpentine, creosote, carbolic acid, quinine, or salol;

(*b*) Making the patient live in an atmosphere saturated with volatile antiseptic substances, vapor of carbolic acid, creosote, oil of turpentine, thymol, compound tincture of benzoin, eucalyptus oil, etc.

(*c*) Directing sprays and vapors of various kinds (similar to the above) into the air passages.

All these methods have been sedulously tried, with greater or less, but on the whole, a very limited and temporary advantage.

(*d*) A method which has proved much more satisfactory, and which is worthy of extended trial, is the *intratracheal injections* of suitable antiseptics. The solution employed by one of the present writers, with remarkable success in several cases, consists of the following combination:

Menthol, ...	10 parts.
Guaiacol, ...	2 "
Olive oil, ...	88 "

of which a drachm is injected through the glottis, by means of a special syringe, twice daily. The result, in all the cases in which we have tried it, was a speedy diminution and disappearance of the fœtor, with improvement in all respects, and that although the usual methods of treatment had failed.

This method of intratracheal injection would seem to the practitioner at first difficult and hazardous, but experience shows that it is absolutely free from danger, and that it may be accomplished with

great ease when applied by one who has had a little practice in the matter. The injection is given as follows: the patient thrusts forward the tongue, grasps it, and holds it there. The nozzle of the syringe is directed over the back of the tongue, care being taken to avoid touching it or the palate, and carried backwards until the tongue and epiglottis are passed. The point is then directed downwards and slightly forwards, the instrument being so curved that with the handle against the upper teeth the point is in the glottis. The injection is then rapidly administered and the instrument withdrawn.

It might be supposed that the laryngoscope was necessary in order to make sure work, but it is not so.

(*e*) Direct injection of antiseptic substances through the thoracic parietes into the cavities. Dr. Mackay employed fifteen-minim injections of a thirty-per-cent. solution of beech creosote in almond oil but without apparent benefit. We have employed this method with a guaiacol and menthol solution in the case of phthisical cavities and with considerable success. But the risk of infecting the pleura has to be kept in mind; and we are not on the whole prepared to recommend it.

Gerhardt recommends methodical expiratory compression of the chest, by which means he thinks he is able to remove the purulent secretion, and so to alleviate the other symptoms. Sometimes also he has used faradism to stimulate inspiration.

Treatment of the Febrile Attacks.

We are often called upon to treat bronchiectatic patients during acute febrile attacks, which, as we have seen, are most frequently due to a sapræmia or a septicæmia, or to septic pneumonia, sometimes to gangrene, or some other complication. The treatment of those febrile attacks, as well as of the complications, has to be carried out on general principles; in addition to which we have to attack the collections of decomposing sputum in which most of the complications have their origin, by the various methods at our command, especially by intratracheal injections of antiseptics; or in suitable cases, as we shall immediately see, by surgical interference.

Surgical Treatment.

Operative interference may sometimes be resorted to with good results.

Since bronchiectasis consists of cavities in which accumulation of secretion and subsequent sepsis occur, it is reasonable to suppose

that, if the ordinary surgical means of incision and drainage can be adopted with safety, good will inevitably follow. That pulmonary cavities may be treated in this way has been proved by experience. Cases have been placed on record where surgical means were adopted to drain the cavities by Williams and Godlee (1886), Grainger Stewart (1887), Barr (1887), Finlay (1888), Mackay (1889), Sutherland (1892); and the experience in these cases seems to warrant similar interference not only in cases where such means as intratracheal injections fail, but also in other cases, and with the hope of bringing about a permanent cure.

The operation is indicated in the following conditions:

(*a*) Where the physical signs point to the existence of one main cavity;

(*b*) Where the physical signs indicate affection mainly of one lung, and especially of one main cavity in that lung;

(*c*) There is no reason why one main cavity in each lung should not be attacked by a double operation, although Godlee and Williams believe that if both lungs are affected it is wiser to abandon all thought of operation;

(*d*) Where all ordinary means have failed, and the patient is in danger of death from septic infection.

The first risk attendant on the operation is that due to septic infection of the pleura. This danger does not exist when, as is often the case, the pleura is firmly adherent over the cavities; and where this is not the case means should be adopted to secure adhesion of the pleura by a preliminary operation of stitching it to the thoracic wall.

The second danger is hemorrhage. This rarely proves formidable at the time of the operation; but it is clear that in any instance a pulmonary vessel might be cut into and formidable hemorrhage might occur. It is not uncommon to find it a troublesome complication twenty-four to forty-eight hours after operation, or even at a later period; and little room is left for more than ordinary hemostatic remedies when it does appear. Local treatment is of course almost impossible.

The operation itself is easy. A hollow needle, connected with an aspirator, is first used to locate the cavity; the needle being thrust in at the point of maximum intensity of the physical signs, and search made until the accumulation of fetid material is thus found. The needle is then used as a guide, along which a free incision is made. It is advisable in most cases to resect a portion of one or more ribs to allow of free drainage; and in certain cases it may be desirable to make a counter-opening. Large-sized drainage tubes

are then introduced, and the cavities regularly washed out with warm antiseptic lotions of suitable strength.

In the first case in which we employed this method, the fœtor disappeared from the day on which the operation was performed, and never recurred. The patient ultimately made a good recovery, and remains to the present time in prefectly good health.

The treatment, therefore, which we would recommend is (1) the use of antiseptics and especially by way of intratracheal injections. (2) Failing benefit from this the question of operative interference must be considered and determined according to the principles already explained.

ASTHMA.

Definition.—The term "asthma" appears to have been originally synonymous with "dyspnœa"; but is at present to be regarded as including only those varieties of dyspnœa due to alteration of the condition of the smaller bronchial tubes of a spasmodic and temporary kind;

Consisting in spasmodic constriction of these tubes with or without accompanying inflammation;

Characterized by the occasional occurrence of paroxysms of dyspnœa due to contraction of the tubes; sometimes associated with local irritation of the respiratory tract, sometimes with inflammatory changes in the tubes, sometimes with nervous conditions influencing the tubes in a reflex way;

Caused by the various conditions above referred to;

Resulting sometimes in complete recovery; sometimes inducing emphysema, collapse, and other pulmonary changes.

History.

A disease presenting the pronounced subjective and objective phenomena of asthma, so sudden in their onset, so urgent during their persistence, and often so peculiar and capricious, could not fail to attract the attention of observers; and so we find it described more or less accurately by the earliest physicians.

The term, which was used by Hippocrates to signify "hurried breathing," is derived from the Greek word ἀσθμαίνω, "I gasp for breath;" and it is to be remembered that not only by Hippocrates himself but also by all writers up to the end of the seventeenth century, the word is used in its etymological sense, and is applied to all kinds of disease of which "gasping for breath" is a leading symp-

tom. But the "gasping for breath" of the disease to which alone the term asthma is now properly applied is so distinctive and peculiar, that notwithstanding ignorance of the methods of physical diagnosis, and crude notions of pathology which made a correct differentiation between diseases and symptoms impossible, we find in the old writers graphic pictures of the clinical aspects of asthma and even views as to its nature which foreshadowed modern theories.

From the end of the seventeenth century onwards asthma came gradually to be regarded as a distinct and independent malady, v. Helmont [78] and Willis [79] having discovered that some people suffered from peculiar attacks of shortness of breath, and that after death their organs and especially their lungs were found healthy—*viscera omnia sana præsertim pulmones.*

We find Sir John Floyer [80] in 1698 writing an interesting "Treatise of the Asthma" in which he tells how he himself "had suffered under the tyranny of the disease for at least thirty years;" and he, an acute observer, gives a vivid account of the asthmatic paroxysm, and of the many causes which in his personal experience and among his patients he had found to induce the attack. Anticipating more favored pathologists of later days he assigned as the immediate cause "straightness or constriction of the bronchia."

Little, however, was accomplished toward accurate separation of asthma from other diseases attended with dyspnœa until the time of Laennec,[3] when the use of percussion, auscultation, and other methods of clinical diagnosis introduced scientific precision into medicine and new life into pathology. For although Brée [81] put forward a theory which has to this day not a few advocates, namely, that the primary cause is an exudation into the bronchi, and that the dyspnœa of asthma is due to an effort to expel irritating material, his theory must be regarded as of equal value with those which he tried to refute, since he was unable to distinguish between the dyspnœa due to asthma and those due to such maladies as uræmia and œdema of the lungs. Laennec, on the other hand, as a result of his investigations with the aid of the new methods, was able to prove that many supposed cases of asthma were in reality due to gross and permanent cardiac and pulmonary lesions—discoverable during life and demonstrable after death—of which shortness of breath is one of the symptoms; and that an affection characterized by paroxysmal attacks of dyspnœa, not associated with demonstrable organic lesion, and leaving in the interval between the paroxysms no trace of its presence, must be distinguished from these, and that *it* constitutes the disease properly called asthma. He reasoned from what his stethoscope told him, and from the negative results of post-mortem examination, that

the immediate cause of the affection must be a spasm of the muscles of the bronchi which had been discovered by Reisseissen.

The use of the new methods of diagnosis in the hands of Laennec may therefore be said to have established the fact of the existence of the idiopathic affection called asthma; for although some early observers, notably Rostan, Louis, and Beau, were, according to Trousseau,[82] at first inclined to deny the existence of an independent disease, and found in catarrh of the lungs, emphysema, and so forth, satisfactory explanations of what they claimed was only a symptom, the fact of the existence of asthma as a distinct clinical entity has since that time been universally acknowledged.

This recognition of asthma as an independent disease became, however, the starting-point of a long controversy as to its exact nature and pathology. Old theories were revived and rehabilitated, and new theories were invented to account for phenomena so strange and so interesting; and even at this day there is by no means any absolute unanimity as to the essential cause or the essential nature of the disease.

Without entering into an account of the early part of the controversy; of the physiological researches of Reisseissen[83] and of Kölliker[84] into the histology of the bronchi, and the existence of muscular fibres in their walls; or the experiments of Williams[4] and of Longet,[85] of Volkmann[86] and of Paul Bert[87] on the bronchial contractility (which went finally to prove the existence of muscles, even in the minutest bronchial ramifications), we may enumerate the views which have a practical interest at the present time.

1. The theory which is maintained under various modifications by the largest number of observers, and supported by the most convincing clinical and pathological phenomena, is the old theory foreshadowed by Willis,[79] by Floyer, and by Cullen,[88] the theory of Laennec and of Trousseau, that asthma is essentially a neurosis involving the respiratory centres or their afferent or efferent nerves, inducing spasm of the bronchial muscles; the paroxysm, and perhaps the whole morbid process being frequently induced by inflammatory and congestive conditions of the respiratory tracts, and particularly of the bronchi, also by peculiarities and idiosyncrasies in relation to certain substances, by irritation propagated from the nerves of the gastric and intestinal mucous membranes, and of many other parts; but also dependent on simple irritability of the respiratory centres, and sometimes on mechanical irritation of the afferent or efferent nerves.

This was the theory of Laennec, who, while attributing some cases of asthma to dry catarrh, concluded that the most frequent

cause was a spasm of the muscles of the bronchi and of those of the alveoli where he erroneously thought they existed.

This was also the view of Andral,[89] who found in an abnormal irritability of the vagus and phrenic nerves the cause of the bronchial spasm.

Ramadge[90] accepted the nervous theory as also did Romberg,[91] and Bergson[92] in an able work published in 1840 drew a clear distinction between idiopathic nervous asthma, and those other forms of dyspnœa which occur in the course of gross pulmonary and cardiac lesions. Asthma, according to him, is an independent neurosis caused by either central or peripheral irritation of the nervous mechanism of respiration.

Trousseau regarded asthma as a diathetic neurosis, and the paroxysms of dyspnœa which characterize it as probably the result of a spasmodic constriction of the bronchi, which by narrowing for a time the calibre of these tubes prevents the free circulation of air in the lungs, and gives rise to all the phenomena which follow.

Hyde Salter,[93] in his great work on asthma published in 1860, advocates the same view, and adduces many interesting clinical facts in its support.

Biermer[94] in 1870 regarded asthma as essentially due to spasmodic contraction of the bronchial muscles which render inspiration and expiration more difficult, the disturbance of expiration being, however, more important than that of inspiration, as the sphincter-like occlusion forms a barrier more difficult to overcome in expiration than in inspiration. Bronchial hyperæmia he thinks a consequence of the asthma, as Trousseau had before him maintained.

Other authors as Lebert[9] and Riegel,[10] Leyden[95] and Curschmann[96] give the bronchial-spasm theory a modified support.

And in more recent times this theory has received a new and remarkable confirmation in a new direction. Prof. T. R. Fraser,[97] taking as his basis the well-known pharmacological action of the "nitrite" series of drugs, namely, the relaxation of the spasm of non-striped muscle, conducted a series of clinical experiments in order to ascertain what influence these bodies exerted on the dyspnœa of asthma and of bronchitis. His valuable observations afford a convincing proof of the correctness of the theory of bronchial spasm as the cause of asthma, and tend also to show what is of considerable interest in relation to treatment, that an element of spasm enters into the dyspnœa of ordinary bronchitis.

2. Others have maintained that a spasmodic contraction of the diaphragm at its maximum of depression affords the explanation of the phenomena. The modern champion of this theory is Wintrich,[98] who, basing his position on the physical signs found during the

asthmatic paroxysms, and on experimental investigation, argues that a nervous asthma is impossible as a result of spasmodic contraction of the smooth muscular fibres of the bronchi. The low position of the diaphragm he thinks is incompatible with spasm of the bronchi; nor can enlargement of the lungs with increased air contents be explained by a spasmodic contraction of the bronchial muscle. On the other hand, he thinks that all the phenomena can be easily explained by a tonic spasm of the diaphragm either alone or combined with spasms of the other respiratory muscles.

Wintrich in this contention received the support of Bamberger[99] and Lehmann,[100] while Lebert[9] and Jaccoud,[101] though admitting the importance of the spasmodic contraction of the diaphragm, thought spasm of the bronchial muscles also a necessary element. Budd,[102] Kidd,[103] and Germain Sée[104] advocated a different view in that they believed that all the inspiratory muscles, and not the diaphragm alone, were in a state of spasm, and that the spasm might be due to irritation of the vagus, either peripheral or central. Sée's position, inasmuch as he clearly expressed the opinion that the central seat of the affection is in the medulla oblongata, and the essential nature of the disease a neurosis of that centre, might more correctly be indicated by placing him in our first group.

3. Some have thought the process due to sudden turgescence of the bronchial mucous membrane. Brée, ignorant, however, of the true limitations of the disease, had already put forward this theory and it has become revived in several forms.

(*a*) Traube[105] held the same opinion as Wintrich in regard to contraction of the diaphragm; but in his opinion the cause of such contraction was an effort to remove an obstacle in the air passages. This obstacle he thought at first a result of a catarrhal, but afterwards of a vaso-motor disturbance.

(*b*) Weber[106] in 1872 and Störck[107] in 1875 were not able with the theory of bronchial or diaphragmatic spasm to explain all and especially the auscultatory phenomena of bronchial asthma. They, on the contrary, regarded the disease as essentially a vaso-motor neurosis, causing sudden and abnormal swelling of the bronchial mucous membrane by overfilling of its vessels, the disturbance of respiration being secondary to the vaso-motor congestion.

(*c*) Sir Andrew Clark[108] referred the process to a change in the mucous membrane resembling urticaria of the skin, and which came and went as urticaria does.

(*d*) Berkart[109] in a recently published work revives the catarrhal theory, and maintains that the turgescence is one of the phenomena connected with a diffuse erysipelatous inflammation of the mucous

membrane, due to the action of a streptococcus, and starting invariably from the pharynx.

4. Some observers have conceived that morbid conditions of the blood, influencing at one time the skin, at another the mucous membrane, afforded the best explanation. Early in the century Duclos [110] spoke of a herpetic tendency of this kind, while Trousseau thought the neurosis essentially diathetic, and like Murchison [111] attributed to gout a share in the production of the disease. Trousseau warns his pupils to remember "that eczematous and rheumatic affections, gout, gravel, hemorrhoids, periodic headaches, and asthma, which are varying expressions of one and the same diathesis, may replace one another."

5. Certain observers attached great importance to the occurrence of some specific products which acted as local irritants to the bronchi and produced the symptoms. Among these may be mentioned the Charcot-Leyden [112] crystals, the spirals of Curschmann,[96] the minute casts or moulds of bronchial tubes, and the eosinophile cells [113] found in the sputum of asthmatics. These crystals, spirals, and eosinophile cells have been supposed to act as irritants and directly or reflexly to produce the symptoms.

6. The starting-point of the asthmatic paroxysm has been sought not only in the bronchi, but also in other portions of the respiratory tract. Affections of the nose have in this connection received much attention since Voltolini [114] published a case in which asthma was cured after the removal of a polypus.

Voltolini's experiences have been confirmed by several workers in this department—by Haenisch,[115] Hack,[116] J. N. Mackenzie,[117] and Schmiegelow.[118] More recent observations have also established the fact that irritation of the mucous membranes of the pharynx and larynx may induce asthma, as has been shown by Thornwald,[119] Brügelmann,[120] and others.

Schmiegelow, from a careful study of a large number of cases, comes to the conclusion that asthma must be considered a bulbar neurosis, which consists in an excessive reflex irritability of the respiratory centre; that an asthmatic attack in many cases may originate from the mucous membrane of the nose if only the necessary morbid condition as well as the increased bulbar reflex irritability be present, just as, *cæteris paribus*, irritations conducted from any sensory nerve whatever are capable of causing asthmatic attacks.

Pathology.

There is no lesion discoverable after death which can be said to be essentially characteristic of asthma. For as we believe, asthma is

a neurosis consisting in subtle changes in the central nervous system not recognizable by our present methods of investigation—a conclusion to which we are forced not only by the clinical features of the disease, but also by the very absence of such morbid changes in the lungs as could be supposed to be capable of producing the phenomena.

It is true that alterations may frequently be found in the lungs and other organs which have a relation to asthma. But these are the results, not the cause of the asthmatic attacks. Asthmatic patients are, probably on account of the nervous irritability of the respiratory organs, liable to vaso-motor congestive and catarrhal affections of the lungs, which by frequent repetition induce the chronic catarrhal changes which are the lesions oftenest met with in the bodies of those who have long suffered from the disease.

It is easy to understand also how the oft-repeated recurrence of attacks of severe dyspnœa leads to changes in the lungs of the nature of emphysema and collapse and secondarily to hypertrophy and dilatation of the right side of the heart and other sequelæ.

According to Geddings,[121] a frequent change in the bronchial tubes of old asthmatics is hypertrophy of the circular muscular fibres, causing thickening of their walls and diminished calibre—a condition which is also described by Williams.[122]

The bronchial glands may be found enlarged. It is said that asthma not infrequently follows on arrested phthisis in adults, from enlargement of the glands pressing on the vagi.

According to Hack,[123] the nasal condition which has a special causal relation to asthma is hypertrophy of the vascular tissue on the anterior end of the inferior turbinated bones.

Berkart[109] has made a statement which we are not able to confirm, that the epithelium is the starting-point of the morbid action; that during an attack it exfoliates from the greater part of the respiratory tract and especially from the smaller bronchi, and that it undergoes peculiar changes which, with fibrinous exudation within the bronchioles and alveoli, constitute the casts and spirals found in the expectoration. These are supposed to act as emboli, and by obstructing the bronchial tubes to cause the disturbance of respiration. He attributes the whole process to the action of a streptococcus.

Clinical History.

Asthma may be described as presenting various types, namely:

1. Hay asthma and allied processes.
2. Pure spasmodic asthma.
3. Bronchitic asthma.

1. *Hay Asthma.*—Some individuals exhibit an abnormal sensitiveness of the respiratory tract to certain sources of irritation—a sensitiveness which may be described as an idiosyncrasy, inasmuch as the vast majority of people are not in the slightest degree susceptible, although exposed to the same influences. The most notable source of irritation of this is the pollen of grasses, which has been proved to be the exciting cause of a disease prevalent in Britain and elsewhere during the grass-flowering season, and known as hay fever, of which hay asthma is the most important symptom.

1. *Hay fever* exhibits three clinical sub-varieties:

(*a*) That in which the nose and eyes are specially affected;

(*b*) That in which the bronchi are specially involved;

(*c*) That in which the two are associated.

(*a*) The disease sets in with some malaise and lassitude, and a feeling of tightness and oppression about the head, accompanied by itching of the eyes, chiefly in the region of the inner canthus, and of the alæ of the nose, and a prickling sensation in the throat. These are speedily followed by those symptoms which give the disease its distinctive character. The itching gives place to great uneasiness, the conjunctivæ become red, the nostrils pour out a profuse thin watery secretion, and the tears overflow. The eyelids, nose, and cheeks become irregularly swollen and congested, so that the patient looks like one who has had a fit of crying. Violent, prolonged, and frequently repeated paroxysms of sneezing set in, and the patient is obliged to sit sneezing and attending to the flow from his nose all day. At the same time that the nostrils pour out their secretion almost in a constant flow the mucous membrane becomes so swollen as to occlude the nasal passage—and this interference with nasal respiration adds to the patient's misery. Taste and smell are in abeyance.

These symptoms last for days or weeks and reduce the sufferer to a condition of extreme exhaustion and collapse. They are subject to aggravation if the patient ventures outside, or into the country, or if the days are bright and sunny. A wet day, on the other hand, brings comfort, the source of the peculiar irritation being laid by the rain.

(*b*) These are the common features of hay fever, and they suffice to make the sufferer miserable enough. But frequently a more serious and distressing element is added when the bronchial mucous membrane is specially or almost exclusively involved.

The bronchial mucous membrane may exhibit a special sensitiveness to pollen in those who are already asthmatic: while in others pollen is the only irritant which excites asthma so that their disease is truly a hay asthma. At the particular period of the year—in our climate in the latter end of May and throughout June—when the

grasses bloom and hay-making goes on, the patient who is thus affected never knows comfort, if he is in the neighborhood of hay-fields or if the breeze blows toward him over grassy meadows. At any moment he may be seized with a severe paroxysm of urgent dyspnœa with cough and tightness of the chest, and the loud wheezing and other signs of asthma. In some cases there is a profuse mucous expectoration.

These symptoms last with recurrence and exacerbations so long as the exciting cause is in the atmosphere. Patients indeed know from experience when they will come and when they will go. Many can forecast almost the exact day on which the old enemy will return, and contrive to escape from its tyranny by opportune seclusion, or migration to the sea-side as much removed as possible from the sphere of its influence. Occasionally special sensitiveness to other impressions is met with, particularly in the bronchial tubes. Plants such as privet (*Ligustrum vulgare*), the powdered root of ipecacuanha, various scents, the scarcely perceptible odour of a cat or a piece of fur, suffice to induce distressing and sometimes prolonged attacks.

(*c*) In a number of cases the nasal and the pulmonary symptoms are associated. Very commonly in these cases the nasal symptoms precede the pulmonary by a few days, so that we get frontal headache, streaming eyes and nose, and paroxysms of sneezing, followed and complicated by, sometimes replaced by, paroxysms of urgent dyspnœa with profuse mucous expectoration. The bronchial mucous membrane, protected so long as the respiration is nasal, becomes exposed to the same irritant when the nose becomes blocked and the breathing is carried on through the mouth.

2. Pure spasmodic asthma, or *bronchial asthma*, includes all the forms of asthma in which the condition manifests itself without any prominent bronchitis and without special sensitiveness to pollen.

The disease in its uncomplicated form has two outstanding characteristics, the occurrence of more or less frequent paroxysms of a peculiar dyspnœa, and the complete absence of all symptoms or traces of the disease in the intervals between the attacks.

The paroxysms of dyspnœa or the "fits of asthma" may come on with alarming suddenness, or may be ushered in by some prodromal symptoms, which are intelligible warnings to the old asthmatic, and which by those who maintain the neurotic origin of the disease have sometimes been compared to the "aura" of epilepsy. These symptoms are not infrequently alimentary, such as great flatulence; or nervous, as sometimes general irritability with various hyperesthesiæ, but more commonly great drowsiness and languor; or urinary, as profuse diuresis.

With or without such prodromal symptoms the attack comes on. It occurs most frequently during the night, awaking the patient out of a sleep into which he may have fallen feeling quite well, but which is often disturbed by dreams and nightmares of something pressing him down and weighing heavily on his chest. He awakes to the reality and to an anxious struggle for breath. There is a feeling of great general oppression, and of tightness of the chest and inability to expand it. His breathing is slow and labored, and accompanied by a loud wheezing and rattling. He is obliged to sit up, he clutches at anything by which he can fix his arms so as to bring into action every muscle of respiration. The face, at first pale, becomes livid and turgid, the eyeballs protrude, and as the attack continues, the cyanosis becomes extreme. Sometimes the deficient aëration of the blood gives rise to an instinctive desire to throw a window open and to put out the head into the cool air; sometimes in the struggle for breath the patient assumes attitudes which enable him to bring the auxiliary muscles into play. The body is bathed in a clammy perspiration, the extremities are cold; the heart's action is disturbed by the great strain put on the right side; its action becomes sometimes tumultuous and irregular, the pulse at other times is small and feeble.

The attack may last for hours, or even for days, without intermission; it may subside, with the same remarkable suddenness with which it comes on; or relief may come more gradually with the establishment of an expectoration which presents the character immediately to be described.

On examination of a patient during the attack we find the symptoms and physical signs to be as follows:

The attitude is one of orthopnœa, or the patient stands grasping some object and struggling for breath. He cannot speak and scarcely dares to move for fear of increasing his difficulty.

The respirations are slow and labored, as is characteristic of obstructive dyspnœa. And the rhythm is changed so that expiration lasts two or three times as long as inspiration. Both the respiratory acts are performed with a struggle; for the thorax seems to be fixed in a position of permanent inspiration, and neither the powerful inspiratory effort nor the laborious and prolonged expiration make much impression on its capacity. It is commonly stated that the dyspnœa is an expiratory one, because the expiration is prolonged and performed with a manifest struggle; but many patients declare that their main difficulty is inspiratory and that expiration is less distressing. It would seem that with some at least, a tonic spasm of the diaphragm and other inspiratory muscles is superadded to the initial bronchial spasm, fixing the thorax in the inspiratory position, and

that expiration is prolonged because the inspiratory muscles refuse to relax, and the distended lung cannot collapse. It is certain from the fixed position of the thorax, and the slight change, with expiration and inspiration, in the limits of resonance of the lower edge of the lungs as ascertained by percussion, that one of two conditions exists—viz., that bronchial spasm produces an extreme degree of obstruction and especially of obstruction to the egress of air; or that the two sets of respiratory muscles are contracting simultaneously and antagonistically instead of alternately and rhythmically.

The percussion phenomena are those of a temporary pulmonary emphysema. The volume of resonance is increased; the superficial cardiac dulness is diminished or obliterated; the limit of the lower margin of the lung is displaced downward for two inches or more, and is not affected to any appreciable extent by the respirations.

There is a loud wheezing, often more marked in expiration, which is audible at a considerable distance from the patient. On auscultation the normal vesicular murmur is absent or indistinct, being masked by a multitude of sibilations and snorings and rhonchi which are constantly changing their character and situation. The sounds are commonly dry. As the attack is subsiding they gradually acquire a moist character. Vocal resonance is everywhere diminished.

There is sometimes cough; but coughing, like speaking, or any other exertion calculated to increase the respiratory difficulty, is carefully avoided by the patient. As the attack is subsiding, indeed often as the first indication that relief is at hand, the patient begins to cough and to expectorate a sputum which is peculiar and characteristic.

The sputum in an uncomplicated case of asthma consists of a thick, tenacious, glairy, non-aërated mucus, containing transparent pellets and sago-like grains embedded in it. In cases of long standing the expectoration is frequently purulent or muco-purulent.

In this sputum characteristic structures—macroscopic and microscopic—are found. So long ago as 1835 Lefevre [124] (who was himself asthmatic) in a graduation thesis described how he "generally obtained relief after the expectoration of a peculiar kind of sputum of a gray color, tenacious or firm in consistence, filamentous in shape like boiled macaroni, or twisted into spirals, and moulded upon the ultimate ramifications of the bronchi." These naked-eye appearances have been described by many authors in recent years and very fully by Troup [13] and Berkart. They are the appearance of the sputum of an old asthmatic, and the filamentous and spiral bodies are casts of bronchial tubes.

The transparent and sago-like pellets which are expectorated

towards the end of an asthmatic attack are found to consist of the peculiar microscopic bodies now associated with the name of Curschmann.[96] These were first described by Leyden,[112] who found on examination of the sputum in asthma "tubular contorted bodies with oblique spiral lines and a narrow homogeneous-looking thread which runs along their axis and terminates in a spiral twist." These on minuter examination are found to consist of strands and fibres woven and plaited in the most fantastic manner—the plaiting being sometimes very perfect—covered with cells closely packed together, the strands and fibres, coiled or unwound to varying extents, giving rise to the spirals of Curschmann.

Many explanations are offered to account for the occurrence and the structure of these peculiar bodies.

Troup[13] thinks that a considerable portion of the spiral consists of columnar and ciliated epithelium cemented by some fibrinous or albuminous material.

Berkart[109] thinks they arise in the following way: the epithelium of the smaller bronchi is gradually drawn out into fibres like a piece of metal into a wire—the elongation being effected by the drooping of the heavier body of the cell which drags upon its inserted portion. The fibres on opposite sides meeting, interlace and form the plaits; and exudation of leucocytes into the meshes thus formed make up the casts and complete the spirals of Curschmann.

But the appearance of the spirals we have examined suggests a different explanation. For frequently the spirals are directly connected with looser unwoven mucus rich in cells; and one can scarcely look at these without having suggested to the mind the idea that in some way these spirals are produced from the exudation as thread is produced from the loose fibres of which it is composed. The explanation of their production seems to be that certain structures, obviously the minutest bronchioles, during their period of asthmatic contraction seize upon and work into a thread, more or less of the exudative products and mucus which are present in the pulmonary tissue. This at once drives out the moisture and works the inspissated material into the thread-like spirals. Osler[125] has suggested that perhaps the movements of the cilia as well as the muscles of the wall may have a share in the production of the spiral threads, and this may perhaps be the case to some extent.

In the sputum of asthmatics there are also found the microscopic crystals now known as Charcot-Leyden crystals. These are the same as were observed by Charcot[126] and Robin[127] in the blood, liver, and spleen in cases of leucocythæmia, and by Friedrich and Förster[128] in the sputum of bronchitis. Their occurrence in the sputum of asth-

matics was first noticed by Leyden in 1871, who attached to them importance as mechanical irritants to the bronchial mucous membrane. They are minute, sharply pointed octahedral crystals, supposed by Leyden to be derivatives of disintegrating white blood corpuscles. These are found also in the sputum of acute bronchitis; they are identical with crystals seen in post-mortem blood, and also with crystals found in semen.

According to Schreiner[129] they are the phosphate of a base probably identical with ethyl eunim.

The crystals are not found in the sputum of the earlier attacks of bronchial asthma or at the beginning of a fresh attack, but if the sputum is allowed to stand for some time crystals occur in it, and towards the end of an asthmatic attack they appear in the recent sputum.

The sputum also contains a large number of leucocytes showing eosinophile granules. Their significance is not known, but it may certainly be said that those who regarded them as causes of asthma were mistaken.

The severe exertion, the great nervous disturbance and the more or less prolonged defective aëration of the blood produce derangement of the other systems of the body.

During the paroxysm the patient cannot and does not desire to take food; there is great thirst, and frequently there is constipation. The abdomen becomes distended and tympanitic—partly from air swallowed during the paroxysm, but partly also from nervous causes. For this distention of the intestines with gas is a frequent prodromal symptom of asthma, even in cases in which it cannot be attributed to gastric and dietetic disturbance.

According to Berkart "a peculiar odor somewhat like that produced in singeing poultry emanates from the mouth." He maintains that this odor is marked in the acute forms of the disease and is perfectly identical in all cases. We have not been able to confirm this observation.

The pulmonary obstruction caused by the bronchial spasm, together with great muscular effort, leads to backward pressure affecting the pulmonary circulation. The right side of the heart becomes engorged, and the venous return is impeded. There is marked cyanosis, the veins of the neck are prominent, epigastric pulsation is plainly visible, and there is much palpitation.

The superficial cardiac dulness is usually obliterated; the cardiac sounds on the right side may show accentuation; those on the left are feeble, often inaudible. The pulse is accelerated, weak, and frequently irregular, the pulse tension low.

Profuse diuresis is frequently associated with the attacks. But after the attacks the urine is often observed to be diminished in quantity, high colored, and loaded with urates, just as after any other severe muscular effort.

The extremities are often cold; but there is always profuse perspiration. Skin eruptions have been observed in severe cases, such as labial herpes, urticaria, and other eruptions of the sort. But these are exceptional as part of the asthmatic attack. There is always profound oppression and discomfort, often severe headache. Consciousness is seldom lost. Cramps of various muscles are frequent and general tetanic convulsions sometimes occur. The body temperature is lowered.

These symptoms constitute the asthmatic attack. They vary of course in intensity in different cases and at different times; but considering their nature and severity, it is remarkable with what ease and rapidity their effects are recovered from, so that a patient who spends several hours of a night in a violent struggle for breath is able to pursue his avocation in the morning with nothing more than an unpleasant recollection of his sufferings. But many do not escape so easily. The attacks, by reason either of their severity and long duration or of their frequent recurrence, produce such nervous exhaustion and ill-health as to lead to incapacity for work. It is in such cases that the disease merges gradually into the third form.

3. *Bronchitic asthma* constitutes a well-marked variety of the disease, for there are cases of asthma in which the catarrhal element predominates to such an extent as to form one of its main features.

Asthma and bronchitis stand related in various ways. In most cases of nervous asthma towards the close of a paroxysm a hyperæmia of the bronchial mucous membrane supervenes, which may go on to actual inflammation. Therefore bronchitis is the most frequent sequela of asthma. On the other hand, many patients who suffer with chronic bronchitis have from time to time paroxysms of severe dyspnœa which cannot be attributed to an exacerbation of the inflammatory changes, and can only be explained by a superadded spasm of the bronchial muscles. These constitute cases of true bronchitic asthma, a condition frequently met with in hospital patients who suffer from what is known as "winter cough."

We often find that a catarrh starting in the nose spreads along the respiratory tract and involves the bronchi, and there produces symptoms of acute bronchitis. Sometimes in addition severe paroxysmal dyspnœa occasionally supervenes with the characteristic respiration and fixation of the thorax, and such alteration in the physical signs as leaves no doubt that a spasm of the bronchial muscle has been

excited, and has to be reckoned with in the treatment of the condition.

In other cases the bronchitis and the asthma are equally well marked from the outset, so that by some it is held that bronchitis is the sole cause of asthma, and that we never meet with the latter except in association with the former. It is true that in some cases the relation is so intimate that the slightest exposure to cold, inducing a bronchial catarrh, will be the starting-point of a fit of asthma. But in these very cases the amount of bronchial inflammation may be so slight, and the paroxysms of dyspnœa so severe, that we are forced to assume the existence of some other form of obstruction to respiration, which can be none other than bronchial spasm. A perfect analogy is found in the case of the urethra, where a slight inflammation of the mucous membrane, induced, it may be, by urinary irritation, is in certain states capable of producing tight and obstinate stricture—a stricture which no surgical pathologist attempts to explain otherwise than by referring it to spasm of the urethral muscles.

In other cases an asthmatic element complicates bronchitis chiefly of the chronic form. Patients suffering with chronic bronchitis and emphysema are subject to attacks of dyspnœa, which are aggravations of their ordinary condition, and for which no adequate explanation can be found in any increase of the inflammatory process, or any sudden exacerbation of the cardiac inadequacy which so frequently complicates these affections. The behavior of the symptoms—their sudden onset and subsidence, and their recurrence—point clearly to their origin, and a recognition of this element in chronic bronchitis is often of the greatest importance when we come to treat the condition.

Etiology.

1. Age.—Asthma is a disease of all ages. Hyde Salter[99] mentions a case in a baby fourteen days old, and another in one of twenty-eight days; and it may set in in old age, but the majority of cases commence in early life.

Of Hyde Salter's cases 31 per cent. began in the first decade; 12.8 per cent. in the second decade, and 80 per cent. before the age of forty.

According to Berkart's[109] statistics, one-third of all the cases set in the first decade—an experience which coincides remarkably with that of Hyde Salter. By him this preponderance in early life is put down to the tendency which children show to fibrinous inflammation. We think it due rather to the instability of their nervous centres. We have seen cases where the irritation of cutting the

teeth brought on severe paroxysms of coughing associated with dyspnœa of the nature of asthma; and we have seen this tendency disappear as the nervous system became more stable.

2. *Sex.*—By all authorities who have collected a sufficient number of cases to have statistical value it is found that asthma more frequently attacks males than females. This may be due to the fact that men are more frequently exposed to conditions which make great demand on the respiratory organs; for it is established by more than one observer that during the child-bearing years, during which an increased demand is made on the female respiratory organs, cases of asthma in women increase proportionally so as to exceed the number in men of the same ages. Riegel[10] found "that during the period of life in which organic alterations of the lungs are less frequent and in which the nervous system is more sensitive the females preponderate over the males as three to two; while at the period which presents organic disease in increased proportion the males affected preponderate over the females as five to one."

3. *Hereditary Influences.*—Asthma being a neurosis we should expect, from the behavior of other neuroses, to find that the disease is capable of hereditary transmission; and our experience is that the hereditary element is strongly marked, and that, as in many similar conditions, the transmission is more noticeable through the mother to the male offspring. Berkart, in a strictly exclusive table to which he admitted only those cases where the ascendants were under observation, finds a hereditary tendency strongly marked in sixteen per cent. of all his cases.

4. *Social Position.*—Asthma, except as a complication of chronic bronchitis, is not a common hospital disease. It occurs in general among the well-to-do classes—among preachers, lawyers, teachers, and such like.

5. *Surroundings.*—Nothing illustrates the neurotic origin of asthma more clearly than the influence of their surroundings on the individual subject to the disease, which is so great that one writer has remarked that it would seem as if many asthmatics had their own quality of atmosphere, which, and which alone, is with them asthma-generating. The fact that some individuals exhibit an idiosyncrasy to well-recognized irritants which float in certain atmospheres, such as the pollen of grasses or the powder of ipecacuanha, suggests the idea that substances at present unknown which act as irritants to other individuals may exist in the atmosphere of other localities. For it cannot be said that asthma is a disease of any particular country or locality, except, indeed, that it is more common in equatorial regions than in colder latitudes. But, on the other hand, it is certain that

patients exhibit the most peculiar idiosyncrasies in respect of their surroundings. One suffers only in one locality; others suffer when the wind is high, no matter from what quarter it may blow; others, again, are well except when fog prevails. Yet others are ill in clear, frosty weather. Some are miserable whenever they enter a smoky or dusty room, a few have been known whose comfort depended upon their securing a house with chimneys which habitually smoke.

6. Relation to other Diseases.—The relation of asthma to other diseases is a subject worthy of careful study with a view to the elucidation of the true pathology of the condition. We have already referred to the diversity of opinion which exists as to the nature of asthma—a diversity which also manifests itself in the speculations concerning the relation of asthma to other affections.

Thus those who consider asthma a central neurosis point to the recorded alternation of asthmatic and epileptic fits, and of asthmatic attacks with hemicrania and angina pectoris; while those who consider local pulmonary changes to be the essence of the disease give prominence to conditions as antecedent which by the former are regarded as consequences of the attacks.

Pulmonary tuberculosis is said to be an antecedent disease having a causal relation to asthma. It has been suggested by some that the swollen tubercular glands at the roots of the lungs act as irritants to branches of the vagus and so reflexly induce the spasm.

There is a popular belief that those who suffer from asthma are protected from consumption and other maladies, and that indeed asthma makes some atonement for the misery it causes by insuring longevity. But this is unfortunately not true. It is probable, however, that the venous congestion of the lungs which is associated with the asthmatic attacks is unfavorable to the growth of tubercle. In cardiac diseases tuberculosis is rare, and venous congestion is known to be so antagonistic to the growth of the tubercle bacillus that surgeons are becoming accustomed to induce it artificially with the view of checking a tubercular process.

Many find in *bronchial inflammation* not merely a predisposing cause, but also the explanation of all the phenomena of the asthmatic process. Thus, according to Theodore Williams,[122] bronchial inflammation is the cause in eighty per cent. of asthmatic cases. A recent writer (Berkart[103]) goes even further. Dismissing with contempt the commonly accepted theories of the disease as "being based entirely on misconceptions and erroneous inferences," and ignoring the observations of great and trustworthy clinicians, he constructs a fantastic theory by which he attributes all cases of asthma to a fibrinous inflammation involving the whole of the respiratory tract. The patho-

logical process is a progressive form of inflammation of an erysipelatous character, and due to the action of a streptococcus. "Starting, as it invariably does, from the pharynx, it rapidly spreads upwards to the nose and to the eyes; whereas its downward extension is, for a time at least, arrested by the larynx, just as erysipelas is arrested by a projecting fold of skin. That resistance, however, is subsequently overcome and the air passages are invaded." Here fibrinous plugs are formed "which are harmless at the place of their origin"—they do not even excite a cough, but being detached from the bronchioles where they are formed, they get impacted in bronchi of much larger size or even in the main bronchus or trachea, and the asthmatic paroxysm is the expression of the lungs' attempts to remove and expel them.

We have stated this theory for the most part in the author's own words, but we doubt whether it is likely to commend itself to those who have studied the disease. It is to us equally unintelligible that an erysipelatous inflammation can extend from the pharynx by way of the larynx into the bronchi without producing any laryngeal symptoms, as it is that a few fibrinous plugs travelling through tubes of ever-increasing calibre can become so firmly impacted as to necessitate the most violent and prolonged efforts to dislodge them.

By others, again, the dyspnœa of asthma is attributed directly to a sudden increase in the bronchitis which usually causes and accompanies *emphysema.*

Catarrhal pneumonia, whooping-cough, measles, and other diseases which have pulmonary complications, as likewise the acute infective disorders, are said to leave a disposition to asthma. This relation is at least frequently insisted on by the patients or their friends.

The nature of these processes is to our mind such as might very well be the cause of a vulnerability of the parts concerned in asthma whether central or peripheral.

Various *nasal disorders*—chronic catarrhal affections, polypi, and hyperæmias and hypertrophies of special portions of the vascular tissue of the nose—as well as pharyngeal and laryngeal affections, have according to many observers an important share in the etiology of the condition.

Enlargement of the thyroid, congenital or acquired, has for long been observed in relation to asthma, and our recently acquired knowledge of the importance of the thyroid secretion in the economy of the nervous system, as well as the analogy of certain other symptoms connected with disease of the gland, make the frequent occurrence of thyroid enlargement peculiarly interesting. It is certain that the dyspnœa is not produced by pressure of the enlarged gland, as some have maintained.

Certain *general diseases* are known to be so intimately associated with asthma that a causal relation is by many claimed for them. Gout, gravel, rheumatism, rickets, skin diseases such as eczema, urticaria, and herpes—and hemorrhoidal affections belong to this category, and are very properly regarded by some, as for example Trousseau,[52] varying expressions of one and the same diathesis of which asthma is also an outcome.

We thus see that a great many conditions have a relation to asthma, so intimate and so frequent that they have been from time to time put down as causes of the disease. And our conception of the true nature of asthma, as well as our own clinical experience, leads us to acknowledge the correctness of these observations. For there can be no doubt that the true explanation of the symptoms of asthma is a contraction of the bronchial tubes, brought about by spasm of the bronchial muscles, and that the paroxysms of dyspnœa are directly due to the obstruction to respiration resulting from such contraction.

The bronchi are surrounded with a distinct layer of non-striped muscular fibres from their commencement to their termination in the infundibula; and it has been ascertained that these are capable of powerful contraction, influencing in a marked degree the lumen of the tubes. The results of the experiments conducted by Roy and Graham Brown[130] seem to us to afford an interesting confirmation of the prevailing view as to the explanation of the phenomena of asthma; for they found that stimulation of the vagus with an induced current caused powerful contraction of the bronchi of both lungs. After section of one vagus, stimulation of its peripheral end with a current of the same strength caused a much more powerful contraction of the bronchi of both lungs, the tubes appearing to close almost completely. Stimulation of its central end caused a much less powerful contraction; section of the vagus caused a marked expansion of the bronchi of the corresponding lung. Should these experiments be confirmed by other observers it would be proved that the vagi contain for the bronchial just as for the cardiac muscle two sets of fibres—excito-motor and inhibitory—and these must be connected with corresponding centres, which are capable of being stimulated directly and reflexly.

The respiratory centres are not only directly connected with the vagi, which are their excito-motor and inhibitory nerves, but also have sensory communication with the most distant parts. The effect on respiration produced by stimulation of the skin is sufficient to prove this. Doubtless a great deal has yet to be learned concerning these functions, but enough is already known to place the etiology of asthma on a fairly adequate physiological basis. For asthma consists of de-

rangement of the innervation of the bronchial muscles, resulting in irregular and spasmodic contraction of these muscles; and such derangement may consist in an irritability or modification of the respiratory centres, or in irritations applied to their afferent or efferent nerves. Asthma may therefore be purely central in origin, or may have exciting causes in any peripheral part which is capable directly or reflexly of influencing the centre of respiration.

These facts explain what are sometimes called the idiosyncrasies and capriciousness of the disease.

Diagnosis.

We have seen that asthma presents a great variety of clinical types, and that while the disease is essentially a neurosis of the respiratory centres, paroxysms of dyspnœa, which are its principal manifestations, are capable of being induced by a great many causes. Three considerations, therefore, have to be kept in view in relation to diagnosis:

1. The diagnosis of asthma from other forms of dyspnœa;
2. The differentiation of the variety of asthma;
3. The recognition of the direct exciting causes, which is a matter equally of diagnosis and of treatment.

1. The diagnosis of asthma from other forms of dyspnœa. Asthma presents certain positive characters by which it can easily be recognized, such as the sudden and apparently causeless onset of dyspnœa in paroxysms, and the equally sudden subsidence of the symptoms without causing much impairment of the general health. We are guided in many cases also by a history of such attacks recurring with more or or less frequency, and alternating with intervals of perfect health and respiratory freedom.

The character of the respirations is also diagnostic—the breathing labored rather than hurried, and the rhythm altered, expiration being two or three times as long as inspiration.

The sputum is significant, both in respect of its macroscopic and its microscopic features. The sputum which Laennec called pearly, from its occurring in little masses resembling pearls or boiled sago-grains, is very characteristic of uncomplicated asthma.

The physical signs on the part of the lungs—the clear percussion note, the increased volume of resonance, the sibilant and sonorous and ever-changing rhonchi—indicate the nature of the dyspnœa and its immediate cause.

These positive features together with the absence of signs of organic disease of the respiratory, circulatory, and other organs enable us in the great majority of cases to establish a diagnosis.

It is important to remember that asthma is of frequent occurrence in children, in whom the condition is not so easy of recognition. Slight general disturbances are sufficient in them to elevate the temperature, and an asthmatic paroxysm is therefore attended by high temperature; the condition sometimes simulates bronchopneumonia so closely that even an experienced observer like Trousseau has been misled.

The fact that asthma may also be superadded to other respiratory and cardiac diseases must always be borne in mind; its recognition is then not so easy, but it is none the less important especially as regards treatment.

A *differential diagnosis* has to be made between the dyspnœa of asthma and other forms of dyspnœa.

Other Respiratory Dyspnœas.—The dyspnœa attendant on bronchial catarrh and emphysema are of so different a type and pursue so different a course that confusion is not likely to arise. In emphysema there is always more or less shortness of breath, which is subject to aggravation on exertion or from superadded catarrh; and there is abundant evidence of permanent changes in the respiratory and circulatory organs. On the other hand, it is one of the affections in which an asthmatic element frequently plays an important part, the relation being complicated, inasmuch as asthma may be induced by the emphysematous condition of the lungs, while emphysema is one of the most common sequelæ of continued recurrence of asthmatic paroxysms.

Pulmonary embolism occurs in puerperal and cachectic patients, and we usually find evidence of cardiac disease or of thrombosis. Its onset is as sudden as that of an asthmatic paroxysm; its termination, however, is very different. The bloody sputum is an important diagnostic sign.

The dyspnœal paroxysms of spasm of the glottis, as for example in laryngismus stridulus, are easily differentiated by the prolonged and forcible inspiration, the comparatively easy expiration, the laryngeal stridor, and the croupy voice.

Tracheal and bronchial obstruction from any cause—whether stenosis, foreign body, or the pressure of a tumor or aneurysm—may be attended with paroxysms of dyspnœa; but here again inspiration rather than expiration is prolonged; there is stridor, and a peculiar characteristic brassy cough is present.

In paralysis of the posterior crico-arytenoid muscles, there is inspiratory dyspnœa, while the expiration is easy. The fact that the voice is normal might at first be misleading. But the stridor which accompanies inspiration will draw attention to the main air passages,

and a laryngoscopic examination, showing the close approximation of the cords in inspiration, accurately distinguishes the condition.

Cardiac dyspnœa is characterized by rhythmical rapid breathing, the respirations being deep and labored. It is always increased by exertion, and is associated with physical and other symptomatic evidences of cardiac insufficiency.

In the majority of cases there is no difficulty in distinguishing a cardiac from an asthmatic dyspnœa. The cardiac comes on upon exertion, when too great a strain is put on the heart, or on exhaustion, when the heart is unable to continue its ordinary work. This latter fact accounts for the occurrence of cardiac dyspnœa sometimes in a paroxysmal manner. But there are some cases in which apparently true spasmodic asthma becomes superadded to cardiac dyspnœa. The distress then is very great, especially in the early morning. One finds the patient struggling for breath and the respiratory sounds give the characteristic features of asthma proper.

Uræmic dyspnœa is to be recognized by means of the kidney disease of which it is the symptom. For in many instances it partakes of the nature of a true asthma, that is, it is due to spasmodic contraction of the bronchial muscles, induced either by uræmic poisoning of the respiratory centres or by irritation produced during the excretion of the poison by the pulmonary mucous membrane.

At the same time we have seen uræmic dyspnœa unattended by the sibilations and snorings characteristic of asthma, and in which there was simply rapid and labored breathing, with no interval and without any bronchial or pulmonary change. In such a case it appears as if the higher centres for the perception of respiratory needs were unduly irritated, without the irritation extending to the motor nerves, at least of the bronchi.

In *diabetic dyspnœa* there is usually deep and rapid respiration with no interval between the acts, and without any change in the normal rhythm. There is little or no wheezing in the bronchial tubes, but along with rapid breathing there are other tokens of profound disturbance of nerve centres resulting from the effects of the same poison as is disturbing the respiratory processes.

Hysterical Dyspnœa.—In a typical instance of this affection one sees the patient going on, for hours perhaps at a time, breathing with manifest effort at the rate of thirty or more in the minute, but without the slightest sibilant or snoring accompaniment and without any other symptoms, unless perhaps additional manifestations of hysteria.

It is surprising how little the system is disturbed by the rapid breathing, which could not be kept up even by the most resolute person, by voluntary effort, beyond a short time, and even when kept up

for a short time disturbs the action of the heart and the general comfort of the patient.

Spasm of the diaphragm is a very rare manifestation of hysteria. Its symptoms somewhat resemble those of asthma, without these being, however, any physical signs on the part of the lungs.

2. The symptoms of an attack together with the previous history of the complaint, and a careful study of the patient's condition between the attacks, enable us in each instance to distinguish the type of asthma to which a case belongs.

3. It is our duty in every case of asthma to endeavor to discover the direct exciting cause, and we have to recognize that the paroxysms may be excited by a great variety of conditions, as for example:

A. Special sensitiveness of the nature of an idiosyncrasy. This may be exclusively confined to one substance or group of substances, such as pollen or ipecacuanha; and such substance may be the sole exciting cause of the paroxysms, so that if it can be avoided freedom from asthma is obtained, or it may be only one of several causes which are capable of inducing the attack.

B. Irritations may be propagated from various parts of the respiratory tract:

(*a*) Nasal, caused by smells, tumors, polypi, or hypertrophies of vascular tissue; (*b*) pharyngeal; (*c*) laryngeal and bronchial.

C. Irritations may be propagated from the alimentary system, as from the teeth, especially during the second dentition, or from the stomach or bowels.

D. Similarly irritations may be propagated from the various other systems through their sensory nerves.

We have, therefore, diligently to search in each individual instance for any source of irritation, either extraneous, or appertaining to any of the systems of the body, or consisting in a general diathesis such as gout, which can be directly connected with the asthmatic paroxysm. And it may be said that our diagnosis is not complete until that link in the asthma chain is found.

Prognosis.

Although the paroxysms of asthmatic dyspnœa are so alarming to witness, death rarely, if ever, occurs during the fit. Dr. Fagge[131] mentions the case of a student, a strong, healthy young man, but subject to asthma, in whom on one occasion during an attack of the disease an alarming failure of the breathing occurred, the respiration becoming more and more shallow, and eventually ceasing altogether. Fagge thought that, except for the prompt adoption of artificial respiration, the patient would have died.

But such profound disturbance of the respiratory centres is extremely rare. Generally the paroxysm is not attended with danger to life.

Asthma is sometimes completely recovered from. Asthma occurring in children is more favorable in this respect; as the nervous centres become stable and educated, the tendency may completely disappear. Recovery may also follow the removal of a direct exciting cause, such as nasal polypi, or any other known source of irritation for a particular case. Just as a hernial opening will close if the hernia never comes down, so also the respiratory centres will throw off the asthmatic tendency if the special irritation is not for a long period conveyed to them.

Asthma occurring at an advanced age is probably permanent, partly because it is then most frequently associated with permanent organic changes in the lungs.

Long-continued asthma generally produces permanent emphysema and induces chronic bronchitic affections, with all the pulmonary and cardiac sequelæ which result from them.

Treatment.

The treatment of asthma involves the treatment of the patient during the fits and between the fits.

The general indications are—

1. To allay the spasm during the paroxysm;

2. To find out and remove the exciting cause, or to remove the patient from the influence of the exciting cause, if climatic or atmospheric;

3. To treat complications and sequelæ and to improve the general health.

1. Treatment of the Asthmatic Paroxysm.—We have at our command a large number of therapeutic agents which are capable of relieving the dyspnœa and terminating the paroxysm of asthma, and we generally find that each patient has his favorite remedy which experience has proved to him to be efficacious in warding off an attack or in giving him ready relief.

We shall begin with the simplest and least hazardous.

The fumes of burning *nitre* paper, that is to say, of bibulous paper which has been soaked in a solution of nitrate of potassium and ignited after drying, gives complete relief in some, and a degree of relief in a considerable number of cases. Smoking tobacco benefits a few, but the addition of a little stramonium to tobacco, or the smoking of cigarettes composed largely of stramonium is of far greater service. There are many forms of cigarettes sold by the druggists, among which Joys, Savory and Moore's datura tatula, Maori cigarettes,

and various others are much employed in Scotland; while such mixtures as the "Green Mountain Cure," "Himrod's Asthma Cure," and a multitude of others are much vaunted as infallible specifics. Inhalation of chloroform is sometimes followed by marked relief even when used in quantity far short of what would produce insensibility.

Inhalation of nitrite of amyl produces, although in a small proportion of cases, a marvellously good result. We have seen sibilations and wheezing disappear within a minute of its exhibition, and have often heard patients say that they felt relieved by its use, even when the results were less striking. But in a large proportion of cases this, like other remedies, fails to give relief.

Among remedies taken inwardly with good effect, hot strong coffee and caffeine deserve a foremost place, while in some cases an emetic, in others a sharp purge will cut short an attack.

Lobelia, an old remedy for asthma, has fallen into not undeserved disuse. To be of any service it requires to be given in doses which produce very disagreeable, sometimes dangerous, symptoms.

The nitrite of sodium in gr. ij.–v. doses, and nitroglycerin in doses of gr. $\frac{1}{100}$ prove eminently serviceable in certain cases—generally the same as are benefited by the use of nitrite of amyl. The effects of sodium nitrite and nitroglycerin are less rapid, but much more lasting than those of their more volatile congener.

Chloral hydrate in full doses has been found useful, but it is not always a reliable remedy, and sometimes disagrees with patients. Pyridin, although a very offensive drug, has been recommended for use by way of inhalation by Germain Sée,[132] who has also advocated the use of iodide of ethyl for the same purpose, and good results frequently follow the adoption of these methods.

Alcohol is a favorite remedy with some, and often proves useful, especially where there is a cardiac complication superadded, and in such cases also the cardiac tonics, especially digitalis and strophanthus, are to be recommended.

But among all internal remedies that which has proved most serviceable in our hands is the iodide of potassium. We generally prescribe it in the following formula:

℞ Potassii iodidi, ... ʒ ij.
Ammonii carbonatis, ... ʒ i.
Aquæ, ... q.s. ad ℥ iij.
Sig. A drachm in water three times a day, or more frequently as may be required.

It never acts so rapidly as the remedies above mentioned, but it does so within an hour or two of being used, and when it does act its good influence is more prolonged. It is of service in many cases

during the paroxysm; still more frequently is it useful when the attack threatens or when a degree of bronchitis lingers between the attacks. Under the former conditions it is well to give a dose every hour, under the latter three times in the day.

Many other substances could be mentioned which have from time to time found favor as remedies for asthma, such as cannabis indica, grindelia robusta, antipyrin, ammonia vapor, sulphurous acid, conium, hyoscyamus, belladonna, and many more. It is well to remember that different remedies suit different people, and we must attach great value to the patient's experience with various drugs, and adopt the one which suits him best.

We have purposely given the last place to the remedy which is the most infallible in the treatment of the asthmatic paroxysm, namely, morphine administered by hypodermic injection. It gives almost immediate and often lasting relief, but it is a remedy which must be employed with great caution, and should be kept in the physician's own hands and not given to the patient for indiscriminate use; for if the attacks are of frequent occurrence the dose has to be gradually increased, and there is great danger that the morphine habit shall be acquired.

The application of the induced electric current along the course of the vagus in the neck has proved successful in several cases; it is a form of treatment which we recommend as worthy of more extended trial.

The treatment of attacks of hay fever and hay asthma is not very satisfactory. When the disease is distinctly of the asthmatic type the remedies which are found useful in bronchial asthma are serviceable here also. Beyond that, internal medication can be directed only to improving the general health.

The local treatment of the disease is most hopeful when we can discover some morbid condition of the mucous membrane of the nose or naso-pharynx which is amenable to surgical treatment. Although we cannot admit the claim put forward by specialists that some structural or pathological abnormality exists in every case, it is our duty always to look for it. Surgical interference is sometimes attended with the happiest results. This may consist in the removal of polypi, of enchondroses, or of exostoses, or the cauterization of hypertrophied or hyperplastic portions of the mucous membrane. The destruction by the cautery of the whole secretive part of the Schneiderian membrane is a very radical procedure which has been employed in America.

Many local applications are employed: of these, the one which is now becoming very popular is cocaine in solution, either brushed

on or sprayed or sniffed up the nostrils. It is a remedy which we cannot recommend. The immediate relief which it insures soon passes off and leaves behind it an aggravation of all the symptoms. It is, moreover, like morphine, a dangerous drug to place in a patient's own hands.

The inhalation of medicated vapors is of some service in relieving the symptoms. For this purpose many substances may be employed, such as oil of pine and pinol, camphor, tincture of benzoin, carbolic acid, or menthol. Of these menthol is undoubtedly the best.

Dr. Greville MacDonald[1-3] has had excellent results from the use of a solution of chromic acid gr. $\frac{1}{16}$ or $\frac{1}{8}$ to the ounce, sprayed or poured into the nose three or four times a day or oftener, and held in the nose as long as possible.

Ferrier's snuff and menthol snuff give great relief in some cases.

The local application which we have recently found more serviceable than any other is the menthol-guaiacol solution, which gives such good results when administered by intratracheal injection in various pulmonary conditions. It consists of menthol, 10 parts; guaiacol, 2 parts; olive oil, 88 parts; and it is to be brushed on or sprayed into the nostrils several times a day. Patients who have suffered from hay fever and hay asthma for years declare that this application gives more relief than any of the numerous remedies which they have previously tried.

2. *Removal or Avoidance of the Exciting Cause.*—This involves a careful search for any structural or pathological abnormality which may be amenable to surgical treatment, and it embraces also an inquiry into any circumstance which may be found directly or indirectly to have a constant relation to the onset of the paroxysms. It not only simplifies the treatment but often also makes the prognosis more hopeful, when it is possible to make a break in the reflex arc. This may be the avoidance of certain foods, the avoidance of exposure to dust, or pollen, or flowers, or powdered ipecacuanha, or to the effluvia of certain animals, or other specific irritants; it may be the correction of gastric or uterine disorder or a gouty tendency, or it may be the removal of a carious tooth or a nasal polypus.

3. We have *to treat complications* as they arise, and also *to improve by every means the general health.*

Complications and sequelæ must be treated on general principles. We need only mention here that for the bronchitic complications of asthma, no remedy can claim an equality with the iodide of potassium.

One of our most important duties toward our asthmatic patients is to adopt suitable means to maintain and to improve the general health. Such means may be medicinal, dietetic, hygienic, or climatic.

Medicinal.—There are three remedies which are of such signal service in warding off the attacks of dyspnœa and in improving the general health of the patient, that they may almost be regarded as specifics for the disease. These are the iodide of potassium (or such a substitute as the syrup of hydriodic acid), arsenic, and strychnine. These, along with general tonics, such as quinine and iron, improve the condition of the patient, diminish the tendency to the paroxysms of dyspnœa, and influence for good any existing bronchial catarrh.

Dietetic treatment is of the highest importance. Dietetic errors are frequent among all patients; their correction in asthmatics is especially important, inasmuch as the digestive disturbance may be the exciting cause of the dyspnœal paroxysms. So much is this the case that some physicians maintain that "few cases will be found of true spasmodic asthma which are not entirely under the control of well-regulated dietetic management;" and they claim that with a strictly regulated dietary and with sedatives other than opiates between the attacks, permanent cures can be obtained. While all observers recognize that evident digestive disturbances must be corrected, the gastric functions relieved as much as possible, and tendencies to constipation and flatulence guarded against, by some physicians a rule of life as to diet is considered the only certain treatment of the disease. Such rules of life have been laid down by Hyde Salter [93] and by Aitken. The plan recommended by Aitken [134] is briefly as follows: The general secretions are to be attended to for a period of ten days, during which mercurial aperients are given at night and salines in the morning. After these are corrected a strictly abstemious diet is to be commenced, the quantities being regularly weighed out and the hours fixed: (1) Breakfast at 8 A.M. is to consist of half a pint of green tea or coffee with a little cream, and two ounces of dry stale bread. An egg *or* a mutton chop, or cold chicken, or game may be allowed. (2) Dinner at 1 P.M. is to consist of two ounces of fresh mutton without fat or skin and two ounces of dry stale bread or well boiled rice. Beef and lamb are contraindicated, and pork and veal forbidden, as are also cheese and dessert. Three hours after dinner half a pint of weak brandy and water, or whiskey and water, or dry sherry and water, or toast water *ad libitum* may be taken. No fluid whatever is to be taken within one hour of dinner or supper. (3) Supper at 7 P.M. is to consist of two ounces of meat as before with two ounces of dry stale bread. Plenty of fluids—not alcoholic—are allowed, such as soda or seltzer water; but water is the best fluid to drink. This regimen is to be persevered in for a period of at least six months before it can be said, according to these physicians, to have had a fair trial; but a gradual though small addition to the

amount of meat may be allowed after a month, should the symptoms subside and the digestion improve.

It is found by the advocates of this method of treatment that most patients lack the resolution and the self-denial to persevere for a sufficiently long time in a diet so very slender, and we confess that our experience is not such as would lead us to insist upon its adoption in the great majority of cases. It is well, however, to insist in every case upon abstemious living and the avoidance of late meals and heavy suppers; and where a gouty element or other fault is evident such must be corrected by appropriate treatment. But in some cases we find the patients so exhausted, emaciated, and neurasthenic from long continuance of the disease that a liberal though careful dietary, such as is useful in other neurasthenic conditions, has to be adopted.

Climatic.—Reference has already been made to the capricious relationships between certain asthmatics and atmosphere. These render it difficult to establish positive rules. Still on the whole it will be found that the climates which are most favorable to catarrh are best for the asthmatic also. Residence by the seaside or a voyage, or residence in a lofty region, may produce the most salutary results; but the experienced practitioner is always cautious in recommending untried climates to asthmatic patients.

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DISEASES OF THE LUNGS.

(EXCLUDING CROUPOUS PNEUMONIA AND TUBERCULOSIS.)

BY

WINSLOW ANDERSON,

SAN FRANCISCO.

DISEASES OF THE LUNGS.

DISEASES OF THE BLOOD-VESSELS OF THE LUNGS.

In describing the diseases of the blood-vessels of the lungs it is important to remember that there are pulmonary arteries and pulmonary veins, and also bronchial arteries and bronchial veins.

The *bronchial* arteries are the nutrient vessels of the lungs; they frequently vary in number, in size, and in origin. There is usually but one right bronchial artery, which arises from the first aortic intercostal, or by a common trunk with one of the left bronchial arteries. There are usually two bronchial arteries on the left side, which arise from the thoracic aorta, one a little lower than the other. The bronchial vessels are directed toward the back part of the corresponding bronchus along which they run, dividing and subdividing upon the bronchial tubes, supplying them and the cellular tissue surrounding them and the bronchial glands. The bronchial arteries supply nourishment to all the structures of the lungs, while the pulmonary arteries carry in vitiated blood for the purpose of oxygenation.

The *pulmonary* artery conveys the venous blood from the right side of the heart to the lungs. It is a short vessel about two inches in length, arising from the left side of the base of the right ventricle. It passes upwards and backwards to the left side, winding in front and to the left of the ascending part of the arch of the aorta, and divides into two branches of nearly equal size, the right and left pulmonary arteries. The right pulmonary artery is a little larger and longer than the left. It runs nearly horizontally outwards to the root of the lung where it divides into two branches: the lower supplying the lower lobe, the upper supplying the upper lobe and giving a branch to the middle lobe. The left pulmonary artery, shorter and smaller than the right, passes to the root of the left lung where it divides into two branches, one for each lobe.

All these blood-vessels, with certain limitations, are subject to the usual diseases of blood-vessels in other parts of the body. The arteries are subject to acute and chronic arteritis or inflammation,

acute and chronic ulceration, atheromatous, waxy, ossified, and calcareous degeneration, the same as the other arteries in the body.

Diseases of the Bronchial Arteries.

Inflammation.

Bronchial arteritis differs but little from arteritis elsewhere. In the early stage of the inflammation the cellular coat in which the nutrient vessels of the artery itself are chiefly distributed undergoes changes similar in nature to those which we find in inflamed connective tissue in other parts of the body. There is redness from the hyperæmia of the capillary vessels, and swelling in consequence of the inflammatory exudation, but the outer coat and the inner coat in the early stages undergo little appreciable pathological change. As the inflammatory process goes on, the walls become permanently thickened or the inflammation leads to softening and even suppuration of the internal coat, and here is where the real danger of inflammation of the arteries lies. The deposit of fibrin and of inflammatory products generally take place on the inner surface of the middle coat, the endothelium or inner coat of the vessel remaining still intact. Should the inflammation go on to ulceration of the internal coat of the vessel, we are then apt to have embolic clots pass on to be lodged in the lung capillaries. Aneurysmal dilatations may follow at the site of the ulceration, cicatrization and constriction of the arterial wall may also occur, or calcareous and atheromatous degeneration may be set up. For a long time it was supposed that the coagulation of blood or the formation of fibrinous material at the seat of the inflammation was caused by inflammatory exudation taking place from the lining membrane of the vessel. It is now known that the infiltration takes place in the substance of the walls, and that it generally occurs beneath the endothelial lining and breaks through in the case of softening and ulceration. In other words, the inflammation is not usually of the endothelial lining of the blood-vessel but of the vascular layer beneath it. When roughened points from inflammatory deposit appear on the site of inflammation of an artery the fibrin of the blood may coagulate on the spot and in that wise form emboli.

Etiology.

The causes of inflammation of the arteries are varied and many. Acute inflammation may be due to traumatism or some mechanical injury such as the pressure of a tumor, the formation of a thrombus, the impaction of an embolus. Inflammation may spread by conti-

guity from some cancerous ulceration or tubercular formation. Violent exertion, heavy lifting, straining, coughing may induce inflammation of the pulmonary or bronchial arteries. General unhealthiness of the blood, anæmia, scrofula, and syphilis are frequently responsible for inflammation of the blood-vessels. Rheumatism is a very common cause of inflammation of the pulmonary artery, especially in those cases in which there is an endocarditis of the right side. Gout is a frequent cause of inflammation of both veins and arteries, and a cause, too, which we think is frequently overlooked. Inflammation of the blood-vessels is not an infrequent occurrence in low-grade and malignant fevers and the acute exanthemata; in short, inflammation of the arteries of the lungs may be caused by a vitiated condition of the blood acting on the blood-vessels, by morbid deposits in the walls of the blood-vessels, and by causes operating externally on the vessels themselves.

Pathology.

In acute inflammation of the pulmonary and bronchial arteries there is an inflammation of the vascular layer of the vessel next to the endothelial layer. There is swelling of the inflammatory product, which as before explained may ulcerate through the intima or the walls may undergo the various degenerative changes of ossification, calcareous degeneration, etc., depending entirely upon the cause operating in producing the inflammation. As a result of acute inflammation the arterial walls become indurated and thickened; they lose their contractility and power of elasticity. When obstructed by thrombi or emboli they may remain permanently obstructed and may be reduced to mere organized fibrous cords. When ossification takes place they become inelastic, hard, and brittle. When, on the other hand, the walls become softened, aneurysms may form, or ulceration may take place, resulting in perforation and hemorrhage.

Symptoms.

Inflammation of the pulmonary arteries produces symptoms similar to those of general febrile disturbance. There is usually considerable pain on taking a full breath and the pulse becomes full, hard, and bounding. The symptoms often show cardiac complications such as valvular disease, endocarditis, and pericarditis; in fact, cardiac symptoms often entirely mask the symptoms of inflammation of the pulmonary and bronchial arteries of the lungs. We shall speak of thrombi and emboli in another section, and hemorrhage will also be discussed later. Outside of the febrile symptoms, acceleration of the pulse, oppression and pain in the chest on inspiration, with or without the

cardiac complications, there are no very definite symptoms of inflammation of the arteries of the lungs.

Treatment.

Treatment of acute or simple arteritis should consist in remedies which will lower the tension of the pulse, reduce the temperature, slow the current, and relax the blood-vessels. Occasionally it may be wise in full-blooded individuals to practise phlebotomy, taking away from four to sixteen ounces of blood according to circumstances. Otherwise the secret of treating inflammation of the blood-vessels seems to lie in the fact that by dilating the general capillary system as rapidly as possible, we thereby reduce tension in the arteries involved in the inflammation. For this purpose hot baths are employed. Occasionally ice to the chest has proven useful. Minute doses of tincture of aconite, a quarter of a minim every hour, are of value. The administration of liquor ammoniæ acetatis is beneficial. Saline laxatives such as sulphate of magnesium and sulphate of sodium, a teaspoonful of the saturated solution of each given every hour until the bowels operate freely, has proven very successful in the writer's hands. If the inflammation is of rheumatic origin the salicylate of lithium or of sodium would be indicated. Stimulating expectorants, diaphoretics to open the pores of the skin, diuretics to increase the flow of urine, and the salines mentioned, with absolute quiet, rest, low diet, principally milk, will do as much for these cases as can be done. Should the etiology indicate any specific trouble, the treatment must of course be directed to that end.

Chronic Arteritis.

Chronic inflammation affects the pulmonary and bronchial arteries in a similar manner to that observed in other arteries of the body. The degenerative changes which occur are fatty, atheromatous, calcareous, waxy, syphilitic, alcoholic, and gouty.

Fatty degeneration of the arteries is usually a secondary disease. It often goes under the name of atheroma and consists in the early stage of white or yellowish-white spots appearing in the substance of the internal coat or between the internal and middle coats of the arteries. The spots are irregular in size and shape, form elevations and at times ulcerations which encroach more or less on the arterial canal. It seems to be a deposition of fibrous tissue, usually situated external to the tunica intima. This fibrous layer undergoes fatty degeneration. The so-called degeneration is progressive, forming fresh spots here and there, many of which coagulate and form large areas of atheromatous structure. These atheromatous spots are exceedingly

thin at first but increase in thickness and in irregularity as time progresses. The cell proliferation which seems to undergo fibrous formation and then fatty degeneration may soften, become eroded, and gradually disappear, leaving an ulcer-like pit or depression. At times some of the atheromatous material is absorbed and the surface becomes irregularly depressed. As a rule the endothelial lining of the arteries is seldom broken, and is continuous over the atheromatous spots.

Waxy, lardaceous, or amyloid degeneration is another form of disease which attacks the capillaries and the smaller arterioles in the lungs. It is often found in connection with tuberculosis, chronic disease of the bones, amyloid degeneration of the kidney and other organs. The vessels affected become thickened and glossy-looking and assume a peculiar reddish-brown hue when touched with iodine. The waxy degeneration appears to depend on cholesterin combined with albuminous matter. This degeneration is frequently found in systemic dyscrasias such as syphilis, gout, and suppurating diseases.

Calcareous degeneration of the arteries frequently accompanies and often follows atheromatous deposits. Calcareous formation generally commences in the outer part of the internal coat. It forms hard, brittle plates of irregular shape and of various sizes. The calcareous plates are generally separated from the current of blood by a thin layer of shining membrane. Occasionally the erosion of this internal coat takes place at the circumference or edge of the plates and their rough edges become exposed. At times bits of a plate may become detached in the current of blood and thus form emboli. Senile calcareous degeneration of the arteries assumes the form of rings instead of plates. These rings are situated in the muscular coat of the vessel surrounding the entire lumen of the artery. They are frequently observed as annular or bead-like calcifications. These are generally primary deposits and not secondary as is usual with the plates.

Ossification of the arteries is another condition following degeneration. The calcareous matter may be transformed into true bony structure or the atheromatous degeneration may become absorbed and ossified elements deposited in their stead. The earthy matter in calcareous degeneration is deposited in globular masses which soon coagulate, forming plates varying in size from that of a pea to that of an almond. When ossification occurs, true bone corpuscles are manufactured out of this calcareous material, or true bone may be formed without the intervention of atheroma or calcareous deposits at all. The bone plates generally are deposited in the middle coat or between the middle and internal coats, very similar to the atheromatous and

calcareous formations. The result of these different degenerations is that they produce extreme irregularity of the surface; the artery becomes inelastic, more or less rigid; the tunica intima at times ulcerates through; fibrin becomes adherent to the degenerated plates; emboli frequently form in this manner, ulcerations of the vessel often lead to aneurysmal dilatation, and hemorrhage is a frequent result. When any of these degenerations attacks the pulmonary artery the cavities and the valves of the heart are also extremely liable to suffer. These degenerations generally occur in persons of from twenty to fifty years of age. They are more frequent in men than in women, and often seem to depend on syphilis and alcohol for a cause. The annular calcareous degeneration, the so-called senile degeneration of the muscular structure of the tunica media, generally comes on after fifty or sixty years of age, and is frequently associated with gout, rheumatism, or Bright's disease.

Symptoms.

The symptoms due to degeneration of the arteries are not observable for some considerable time. There is little pain and little inconvenience under ordinary circumstances. When there is an ulceration or a fibrous formation within the vessel leading to thrombosis or emboli, the symptoms are more marked. When there is an aneurysmal dilatation or a hemorrhage the symptoms are more apparent. There is generally some roughness of the pulmonary valves and a post-diastolic murmur over the site of the pulmonary orifice. The right ventricle is also prone to hypertrophy and to undergo dilatation.

Diseases of the Pulmonary Artery.

The etiological factors operative in diseases of the pulmonary artery are similar to those affecting other vessels of the body. Gout, alcoholism, syphilis, fatty degeneration of the coats of the artery, general arterial sclerosis in Bright's disease, etc., occasionally affect the pulmonary arterial walls as well as those of the aorta, although to a less frequent extent, as the pulmonary vessel is more deeply seated, more capacious, and less liable to injury or severe strain. Mitral constriction and regurgitation, pulmonary fibrosis, emphysema, hypertrophy of the right ventricle of the heart—in other words, any condition within the heart, the vessel itself, or the lungs may contribute to the degeneration of the pulmonary artery.

Atheroma.

Atheromatous degeneration from the various causes named may attack the pulmonary vessels as well as the aorta. Diseases of the

pulmonary artery before its distribution in the lungs are quite rare. Murmurs most audible over the origin of this vessel are generally regarded as hæmic in origin or functional in character. When neither of these hypotheses is tenable, the murmur is generally attributed to some congenital defect in the heart itself, or to some pressure from without. After all these conditions have been excluded we are still bound to admit that the pulmonary artery may become diseased just the same as the aorta or any other vessel in the body. Post-mortem examinations reveal the fact that the pulmonary artery is not wholly unsusceptible to the ordinary diseases of blood-vessels. Under the combined actions of rheumatism, alcohol, hard work, and syphilitic taint, the pulmonary artery, in common with any and all other vessels, may become the seat of atheromatous degeneration. The atheromatous patches may undergo caseation, ulceration, and osseous new formation. Under such circumstances with great venous pressure and hypertrophy of the right heart, the pulmonary artery may develop aneurysmal dilatations. When there is any obstruction to the pulmonary circulation such as is found in emphysema, chronic cirrhosis of the lungs, chronic bronchitis, bronchiectasis, etc., the right side of the heart at first becomes hypertrophied and later on dilates. Given this hypertrophic condition of the heart with atheromatous degeneration of the vessel, it can easily be understood how it is possible for an aneurysm to develop within or without the pericardium.

Symptoms.—Pulmonary murmurs are systolic in time, soft and blowing in quality, of medium pitch, and best heard over the second and third left costal cartilages near the sternum. The murmurs are generally local, that is to say, are not conducted in any direction from the prescribed area of an inch or two. Murmurs over the pulmonary artery are most frequently heard in young women suffering from anæmia and chlorosis. They are often associated with arterial bruits in the neck and with venous hum over the jugulars. The pulmonary artery as it emerges from the pericardium is very superficially situated, and its orifice is quite close to the sternum, hence it transmits its sounds very distinctly. On making pressure with the stethoscope the sounds are generally much intensified. When there is flattening of the chest walls, such, for instance, as is found in old cases of contracted cirrhosis of the lung, the murmur heard over the pulmonary artery is generally quite intense. Any roughening of this internal surface may be distinguished and it is even possible to diagnose aneurysmal dilatations from those of the aorta, although, as before remarked, such diagnosis is very difficult. On the other hand, aortic aneurysms, enlarged glands, pericardial effusion, tumors of different natures, and even diseases of the lungs themselves may so press upon

the pulmonary artery as to induce murmurs that do not depend upon any disease of the artery itself.

Constriction of the Pulmonary Artery.

Narrowing of the pulmonary artery at its commencement is generally found associated with congenital malformations of the heart, especially with the imperfect septum ventriculorum and patent foramen ovale. Endocarditis affecting the right ventricle may also affect the pulmonary valves and cause them to adhere by their margins so as partly to close the orifice. Myocarditis leading to constriction of the conus arteriosus may also be productive of constriction of the pulmonary artery.

Symptoms.—Diseases of the pulmonary artery near its origin may be recognized by careful auscultation with the stethoscope. There are usually considerable dyspnœa and cyanosis. There may be hypertrophy or dilatation of the heart, and a systolic murmur may be heard over the pulmonary cartilage, conducted slightly upwards and to the left. Narrowing of the pulmonary artery beyond the valves, like other diseases of the artery, is exceedingly rare. The most common causes of such diminution in calibre are attributable to compression by thoracic tumors, aneurysm of the aorta, pericardial effusion, mediastinal growths, and enlarged bronchial glands. When there is an aneurysm of the pulmonary artery or when there is constriction in the pericardial attachment, there is usually a systolic impulse and thrill with great accentuation of the second sound of the heart. There is also present general venous engorgement with regurgitation through the veins in the neck; there may likewise be more or less congestion of the lungs with œdema and generally dropsy. It is claimed that, when there is accentuation of the second sound of the heart, there is stenosis at or within the ventricular orifice.

There is a distinct murmur over the pulmonary artery in many young women and children, especially if they are suffering from anæmia; and the murmurs are frequently associated with arterial bruits in the neck. The exact mechanism of these murmurs has never been altogether satisfactorily explained, but it is believed to depend partly upon the watery condition of the blood and partly upon the fact that the blood does not completely fill the blood-vessel. The murmur may be produced in any blood-vessel by the pressure of a stethoscope so as to interfere with free circulation. In many cases of extreme anæmia these so-called hæmic murmurs may be heard at nearly all of the cardiac orifices. A pulmonary hæmic murmur is nearly always distinctly heard in the back. In marked cases of fibro-

sis of the lung, collapse, atelectasis, chronic bronchitis, etc., the tension of the blood within the artery is always increased. There will then be an accentuation of the second sound. In displacement of the heart from effusion, pericardial or pleuritic, or from tumors pressing upon the heart, various murmurs may be observed.

Diseases of the Pulmonary Artery Within the Lungs.

We have seen that diseases of the main trunk of the pulmonary artery are quite rare, although the artery is undoubtedly subject to atheromatous and other degenerations and even aneurysmal enlargement. The branches of the artery within the lungs are even more rarely affected than is the main trunk itself, with perhaps one exception—namely, in those cases of disease of the lungs in which there are disorganizations of the parenchyma, uncovering the blood-vessels and involving their walls in the destruction, inflammation, and ulceration. It is in the course of the inflammation of phthisical excavations of the lungs that thickening, erosion, ulceration, dilatation, or actual aneurysms of the branches of the pulmonary artery are most frequently met with. Atheromatous degeneration may affect the arterioles, but more frequently we find aneurysmal dilatations on account of the many vessels which are occluded, and the blood pressure, which is as a consequence very much increased. Then there is the loss of support from the degeneration generally of the lung tissues surrounding the arterial walls, and in addition there is an actual softening and degeneration of the arterial walls themselves in the midst of inflammatory changes in the tuberculous cavity. In the acute ulcerative destruction of the parenchyma of the lung, such vessels as generally become occluded by thrombosis are very apt to soften and become eroded, and by their rupture give rise to copious hemorrhages. This, however, is not the most usual cause of pulmonary hemorrhages. In cases in which the destruction of the parenchyma of the lung is less acute, where the cavities form more or less trabeculated walls, the trabeculæ consisting partly of bronchial tubes, connective tissue, and blood-vessels, these vessels are as a rule occluded, but exceptions are occasionally met with in a cavity. It is not infrequent to find in a chronic tuberculous cavity a large branch of the pulmonary artery quite patent, occupying a trabecula or coursing along the wall of a cavity immediately beneath the limiting membrane; indeed vessels may span the cavity entirely. It can readily be understood that such vessels, missing their usual support, become strained by the blood pressure. At first the arterial walls may thicken, but sooner or later an inflammatory process of the nature of an endar-

teritis affects these vessels, and although the walls may be thickened and appear hard and fibroid, they nevertheless yield before the constant blood pressure, producing alarming and at times fatal hemorrhages. Hemorrhages caused by the rupture of those vessels which supply the parenchyma of the lung, which has been broken down and transformed into a tuberculous cavity, are frequently arrested by the thromboses which are set up. In such cases the vessel is occluded as far as the margin of the cavity; here a prominent clot is formed, the remainder of the vessel beyond generally undergoing softening, bulbous enlargement, and degeneration. These plugged vessels gradually undergo severe blood strain, because they have no collateral branches to relieve the circulation, and although originally their walls were thickened, they soon become brittle, soft, and attenuated on account of the bulbous or sacculated enlargement of their extremity. As the aneurysmal enlargement increases, the covering becomes thinner and thinner until any unusual exertion may cause it to rupture. Similar aneurysmal enlargements may occur in the bronchial mucous membrane where there has been erosion or ulceration of one or more coats of the bronchial vessels. Bronchial dilatations may also be the seat of small aneurysms of the pulmonary or of the bronchial vessels.

Treatment.

The treatment of diseases of the pulmonary artery must be largely symptomatic. Absolute and prolonged rest are essential. The etiological factors producing the disease must always be considered. In other words, remove the cause if possible and treat the disease symptomatically and intelligently. In anæmia exhibit ferruginous tonics, taking care that the food is properly digested, and supplying nourishment which can be easily digested. In Bright's disease, address the remedies to the kidneys and to the general arteriosclerosis. In syphilis use the antisyphilitic remedies with ferruginous tonics. In aneurysm restrict the diet and try the iodide of potassium and starvation treatment. In hæmoptysis or severe hemorrhages, hypodermic injections of ergotin, cold applications to the chest, or, what the writer has found very efficacious, equal parts of tincture of opium and fluid extract of ergot in half-teaspoonful doses every hour or two until the hemorrhage stops. In these cases one must avoid stimulants even though the patient is faint, because the hemorrhage stops more rapidly when the heart's action is slow. Sometimes half-teaspoonful or teaspoonful-doses of chloride of sodium in a small quantity of hot water will act well. For the further treatment of hæmoptysis the reader is referred to the article on pulmonary hemorrhage.

Diseases of the Veins of the Lungs.

It is of importance to remember that the lungs have two sets of veins entirely dissimilar in their physiological functions and in their anatomical arrangement. The *pulmonary* veins, which return arterial blood from the lungs to the left auricle of the heart, are four in number, two for each side. The pulmonary veins, be it remembered, differ from the other veins of the body in several respects, namely, they carry arterial blood instead of venous blood, they contain no valves, they are only slightly larger than the arteries which they accompany, and they accompany their corresponding arteries singly and not in pairs. They begin around the pulmonary lobule in a capillary network and are continuous with the endothelium of the pulmonary arterial branches. Occasionally there are three pulmonary veins on the right side.

The *bronchial* veins, on the other hand, return blood from the substance of the lungs the same as any other vein. The veins of the right side open into the vena azygos major. Those of the left side empty into the left superior intercostal or the left upper azygos vein. Inflammation of the veins in the lungs, although of rare occurrence so far as is known, undoubtedly takes place more frequently than we are able to diagnose it. It is a disease of grave importance.

Phlebitis.

The *causes* of phlebitis are almost as numerous as are the causes of arteritis. The veins may be injured and become inflamed. Surrounding structures, tuberculous glands, cancer, aneurysm, and the like may press upon the veins setting up inflammation. Diseases of the blood, such as malignant fevers, small-pox, typhoid fever, and others, may set up an inflammation of the veins followed by thrombosis. Phlebitis may be the result of the absorption of poisonous material from a wound or from infective emboli; erysipelas and other cellular inflammations may extend to the veins and set up serious phlebitis.

Pathology.—The sheath of the vein seems first to become inflamed, especially in that form of phlebitis which spreads from cellular or septic inflammation. The inflammatory process invades successively the outer, middle, and internal coats, leading to their congestion and thickening, and often resulting in suppuration. In the septic variety isolated collections of pus are frequently developed between the coats of the vein, and inflammation may again set up ulceration or erosion from without inwards. In the case of septic material circulating in the blood the internal coat becomes the seat of inflammation, and exu-

dation takes place between it and the middle coat. Thrombosis in such cases frequently occurs and septic suppuration is sure to follow. Occasionally an abscess may open into a vein and convert part of the venous system into an abscess cavity. Pus may be absorbed from a wound or from a tuberculous cavity in the lung and set up inflammation of the veins leading to the heart. Malignant diseases of the lungs frequently set up phlebitis. Syphilitic ulceration of the bronchial tubes, no doubt, causes inflammation of the veins in the vicinity. The diseases which cause inflammation of the cellular tissues generally affect the outer vascular region of the venous walls in a greater degree than they do the inner region, which, it must be remembered, is not supplied with capillary vessels. The walls of inflamed veins frequently become several times thicker than normal; they become congested externally and infiltrated with inflammatory exudation which softens and often undergoes suppuration, in fact, the entire vein may be destroyed by the ulcerative process. At times in chronic inflammation of the veins, the walls having become indurated and the calibre plugged, the whole gradually assumes a fibroid change with the lumen obliterated. There can be no doubt that in phthisical patients tuberculous material is absorbed by the pulmonary veins, which, if carried along, will form thrombi and new foci for the tuberculous ulceration. Phlebitis in the pulmonary veins may also take place in consequence of absorption of septic material from disease of the bones, caries of the skull, in the puerperal state, and from inflamed and varicose veins. The blood in the right ventricle of the heart is sent along through the pulmonary artery into the pulmonary veins, there becomes arrested by the formation of thrombi, and sets up secondary abscesses in the lungs. There is no doubt that many of these embolic or thrombotic clots find their way to the left cavities of the heart and are then sent on to form emboli of either the brain, lungs, liver, spleen, or other organs in the body.

The *symptoms* of pulmonary phlebitis are almost as difficult of diagnosis as are those of arteritis. There are generally symptoms of febrile disturbances, some pain, considerable consolidation about the thrombotic spot with œdema, shortness of breath, cough, and oftentimes hæmoptysis. If the inflammation be septic in character abscesses will form here and there in different parts of the lungs. The constitutional symptoms are generally few, but they are severe from the first; the fever is high, but it soon tends to assume a typhoid character.

The *treatment* consists in perfect quiet, slowing of the circulation, relieving the pyrexia and supporting the system. Little can be done by way of direct medication.

Degeneration.

Degeneration of the veins occurs much more rarely than degeneration of the arteries. Atheromatous and calcareous degeneration have been met with, but as their occurrence is extremely rare we need only mention them.

Concretions.

Phleboliths in the lungs are extremely rare. They have occurred, however, in a number of cases. They lie within the vein, usually enclosed in a distinct capsule. They obliterate the vein in which they are situated, and the distal end of the vein is usually dilated and pouch-like. The bodies vary in size from that of a pea down to that of a bird-shot, occasionally they are much larger. They are of a yellowish-white color, hard and calcareous in nature, formed in concentric layers similar to those of urinary calculus. They are composed of a carbonate or phosphate of lime with some magnesia and organic matter. Their formation is supposed to occur within a thrombus or an embolus, the clot frequently forming a nucleus for the deposition of calcareous material. Once having a nucleus the calcareous salts continue to be deposited in concentric layers.

Growths in the Veins.

Rare circumstances have been observed of carcinomatous and syphilitic growths in the veins of the lungs. These growths obliterate the lumen of the vein and generally set up more or less circumscribed consolidation, fibroid thickening, and eventually ulceration and the obstruction of the lumen. Sometimes secondary cancerous growths originate in the walls of the veins themselves and may form polypi hanging into the cavity of the vessel.

Thrombosis and Embolism of the Lungs.

Definition.—Thrombosis signifies the coagulation of blood in arteries or veins during life. Embolism signifies the transference either of a clot or solid matter, bacteria, etc., appearing within the vascular system, from one part to another in the direction of the circulation.

Etiology of Thrombosis.

Sometimes the coagulation of the blood in a vein seems to depend merely upon stagnation of the blood or sluggishness of the circula-

tion. There seems, however, to be some physiological deficiency either in the blood or in the walls of the vessel which induces thrombosis. The causes may be traumatic, may be pressure from growths, the encroachment of tuberculous cavities, syphilitic ulcerations, the gouty diathesis, or vitiated conditions of the blood. Thrombosis occurs in veins, capillaries, and arteries. The immediate cause of the coagulation is no doubt frequently determined by some roughness of the surface over which the blood passes. This is observed in isolated clots which become adherent to atheromatous patches or ossified arteries. Anything which produces inflammation of an artery or of a vein will also be found to induce coagulation of the blood in the same locality. Anything which alters the normal relations between the walls of the vessel and the blood may cause thrombosis.

Pathology of Thrombosis.

The pathology of thrombosis is essentially formation of a blood clot within an artery, capillary, or vein. The clot consists either of nearly pure fibrin or of all the solid elements of the blood combined. It becomes adherent to the wall of the vessel, corresponding with it in form and mold. The changes which these clots or thrombi undergo in the course of time vary considerably. Usually at first there is a gradual contraction and an attempt at organization, the fluid portion of the clot becomes absorbed, the cellular elements disintegrate and disappear, the fibrinous portion undergoes condensation and in a short while the thrombotic clot becomes converted into firm fibrous or connective tissue, occluding the vessel. Should the clot be septic in character an entirely different change occurs. The centre of the clot instead of organizing begins to disintegrate, there is a small abscess cavity formed, and as the septic material increases, so the clot decreases until a puriform septic mass or granular matter remains. In other cases where there is no septic material in the clot itself, it may begin to soften and break down. The central part becomes converted into thick, puriform fluid and the clot undergoes granular degeneration, leaving cholesterin, white corpuscles, débris, fat globules, and epithelial cells to mark the spot. The fluid portion soon undergoes absorption unless it be septic. The contents become absorbed, the walls collapse, and the vessel becomes obliterated or is converted into a fibrous cord. Sometimes these thrombotic clots become the seat of calcareous transformation. Particles of earthy matter are deposited around the nucleus of some organic matter from the clot. This gradually increases and may be transformed into a calcareous mass of considerable size. Phleboliths are formed in this way and may at times be

the seat of true ossification. Fibrinous clots frequently form in the interior of the right ventricle and extend from the neighboring semilunar valves into the pulmonary artery. Such fibrinous clots, white, firm, and elastic, may certainly be formed during life notwithstanding that many dark, soft, fibrino-corpuscular clots are formed only during the last moments of life. These pulmonary clots may at times be diagnosed many days before death. They always present the impress of the arterial valves. They frequently fill more than half the channel of the pulmonary artery and extend from the semilunar valves into the artery beyond its bifurcation. Cases are on record in which it had been known that these ante-mortem clots were forming in the pulmonary artery for years during apparently perfect health, when all at once, by the shifting of the clot due to some sudden exertion, death occurred instantaneously. The formation of clots in some of the arteries of the lungs is of frequent occurrence in mitral disease and in some forms of obstructive cardiac disease. Thrombosis is a constant accompaniment of pulmonary apoplexy and no doubt in many cases precedes it. After the impaction of an embolus there is nearly always more or less inflammation about the clot, and a thrombosis is formed extending some considerable distance beyond the embolus. In puerperal women, in pyæmic processes, in conditions of diseased bone, tubercular ulcerations, syphilitic disease, and a low vital condition generally, general thrombosis is not infrequent in the pulmonary circulation, causing death almost instantaneously. Many of these cases may be due to the conveyance of emboli to the lungs where thrombosis occurs immediately, resulting in acute congestion, œdema, and a blocking of the circulation, overcoming the power of the heart, and resulting in acute dilatation of the right cavities and death. Pulmonary thrombi in the larger branches of the pulmonary artery occasionally exist in the course of low-grade fevers. There are several cases on record in which well-formed fibrinous clots nearly filled many of the larger pulmonary arteries, showing that they must have been formed some considerable time prior to death.

Etiology of Embolism.

This depends upon any condition which will produce a foreign body or a coagulum within a blood-vessel that may be transferred and lodged in some other vessel, such, for instance, as one of the pulmonary ramifications in the lungs. An embolus may be composed of particles of coagulated blood or of some solid material, such as fibrin, derived from the blood, or it may consist of calcareous and atheromatous particles detached from the interior of some blood-vessel. Septic

material may enter the blood and form emboli in the lungs. In the puerperal state septic micro-organisms may be absorbed by the uterine cavity and find lodgment in the pulmonary branches. Emboli of the bronchial arteries may arise from vegetations from the valves of the left side in the heart. Vegetations of the valves on the right side of the heart, if carried along by the circulation, will of course produce embolism of the branches of the pulmonary artery. Organized fragments may be swept on from aneurysmal dilatations. A bubble of air has been known to produce an embolus. After an injury fluid fat has been absorbed, entered the circulation of the lungs, and produced death. Infective emboli are metastatic in nature and set up suppuration not only in the lungs but in other organs of the body. Ulcerative endocarditis is a frequent cause of pulmonary emboli. Coagulation of the blood may occur in the heart and at the entrance of the pulmonary artery, as we have seen in thrombosis. Particles of this may be swept on by the blood current and produce emboli in many branches of the pulmonary artery. In phlebitis of the lower extremities clots become dislodged, are swept away to the heart, and ultimately become fixed in some part of the arterial system. In rheumatism of the heart, endocarditis, and valvular affections, the lungs do not suffer so much from emboli, because the left cavities of the heart are most frequently affected. But in malignant endocarditis or septic endocarditis due to poisoning of the blood from some septic absorbing surface, the right side of the heart is usually the one affected. Hence embolism and thrombosis are frequent causes of death when the right heart is affected. When an embolus of whatever nature is swept along the blood current until it reaches the final ramifications of the pulmonary artery or until it enters one which will no longer permit it to pass, it becomes a plug and obstructs the blood current in that vessel. Soon after the arrest of an embolus thrombosis occurs and the embolus becomes invested in a clot. The embolus causes coagulation on its proximal side as far back as the next branch given off from the obstructed artery. There is generally also coagulation or thrombosis beyond or on the distal side of the embolus. As the clot solidifies, the watery portion of the blood will be absorbed and the purely fibrinous material remains. This soon undergoes degenerative changes and gradually becomes organized, if it be not septic, and permanently occludes that branch of the blood-vessel. In septic embolism and the embolism from micro-organisms there is also a thrombotic clot formed, but instead of organizing it soon undergoes putrefactive degeneration, forming a pus cavity instead of fibrinous obliteration of the artery. This infective material rapidly ulcerates

through the walls of the vessel and forms pyæmic and embolic abscesses throughout the substance of the lungs.

Symptoms.

In the formation of thrombi the result is not accomplished as rapidly as in the case of emboli. In both cases the vessel becomes obstructed and distended; in both cases there may or there may not be inflammation; in both cases there may be thickening and organization of the clot with obliteration of the vessel, but in the case of the thrombus the process is slow; and it may take several hours or days completely to occlude the blood-vessel, while in embolism the process is rapid. The blood current sweeps the embolic particle on to its final destination, and there it stops suddenly, producing more or less shock, pain, and the immediate symptoms of impeded circulation. It will be remembered that in their final distribution the pulmonary arterioles do not anastomose but form continuous channels around the lobules with the pulmonary veins, so that when an embolus or a thrombus affects one of these final branches of the pulmonary tree there is complete cessation of circulation beyond the obstructed spot. This then would be termed an *infarct* and generally has its base directed toward the pleural surface. The nutrition of the territory supplied by this arterial twig becomes disturbed. If the occurrence is only occasional the symptoms are not so pronounced, as it merely closes certain portions of the lung or produces consolidation and pneumonic spots in their immediate vicinity. But when there are a number of embolic infarcts or a number of pulmonary thromboses going on more or less at the same time, setting up consolidation, inflammation, suppuration, and molecular death or gangrene of the parts, the symptoms are very grave and death generally ensues in a few hours. When an embolic clot in phlebitis, puerperal fever, or pyæmia enters the right cavities of the heart and attempts to pass the semilunar valves, death may occur instantaneously from acute dilatation or spasm of the heart. The symptoms indicative of serious obstruction to the pulmonary circulation are sudden embarrassment of respiration and great dyspnœa, the face assumes a deadly pallor, the skin becomes wet and clammy, huge beads of perspiration stand out all over the body, the pulse becomes feeble, rapid, and irregular, and death may result within a few seconds or within several hours or days after the attack, the time varying according to the size of the vessel obstructed and the area of lung affected.

Treatment.

The treatment must be entirely symptomatic; restoratives must be at once resorted to in serious cases. Hypodermic injections of strychnine, ether, digitalis, and whiskey should be given. The patient should assume a horizontal position with the head slightly elevated; warm cloths should be placed over the chest and warm bottles around the body. Absolute quiet must be observed. Transfusion of blood or a saline solution may at times be tried with benefit. In cases of phlebitis of the extremities great care should be taken that the thrombi are not dislodged and thus sent to the heart. In the case of puerperal sepsis the cavity of the uterus should be carefully cleansed and treated antiseptically. In the case of erysipelas, diseases of the bones, etc., great care must be exercised that septic material be not absorbed. If the disease progresses and abscesses form in different parts of the lungs the case must be treated on general principles. Should the disease progress for several days great care must be taken to support the system with appropriate tonics and nourishing food.

Aneurysm of the Pulmonary Artery.

Aneurysm of the pulmonary artery within the pericardium occurs but rarely. Occasionally there may be found an aneurysm of the pulmonary artery outside of the pericardium. It occurs as a pulsating tumor in the second left intercostal space, with a distinct blowing aneurysmal sound shortly after or continuing through the second sound of the heart. It is almost impossible during life to distinguish pulmonary aneurysm from aneurysm of the arch of the aorta. Occasionally small aneurysms develop in the lungs, the branches of the pulmonary arteries being usually involved. Sometimes a small branch of the bronchial artery situated in the submucous tissues of a bronchial tube may be partly injured by an ulceration and develop an aneurysmal dilatation. The formation of aneurysms in the lungs is seen most frequently in connection with tuberculosis. The reason why hæmoptysis does not occur more frequently in connection with diseases of the lungs and bronchi, in which there is ulceration and destruction of tissue, is because the vessels leading to the spot where the ulceration or destructive inflammation is in progress become thrombosed, and the result is a plugging of their lumen and the prevention of hemorrhage. But in connection with the formation of tuberculous cavities in the lungs the tissues undergo congestion, inflammation, suppuration, and the pus usually finds its way out of the cavities thus formed through one or more communicating

bronchi. In the formation of these cavities the blood-vessel seems to be the last to suffer, as we frequently find a blood-vessel stretching across a cavity. In that event, the vessel, having lost its support by the caseation and expectoration of the surrounding tissues, is apt to dilate, forming an aneurysm. The more usual, however, is the formation of small aneurysmal bulbs near the margin of the tuberculous cavity. The blood-vessels in the process of the destructive inflammation become thrombosed, as before mentioned, and those passing through the abscess cavity also undergo degeneration and are expectorated along with the other pulmonary tissues, leaving the vessel plugged at the surface of the cavity. The plug becomes absorbed more or less, excepting right at the end of the ulcerated vessel, and as the blood continues to be forced into this plugged end, bulbous dilatation usually supervenes. As the diseases progress the cavity enlarges and the destructive process continues. These aneurysmal bulbs frequently rupture, giving rise to serious, alarming, and at times fatal hemorrhages. During life it is impossible to detect these minute aneurysms in the tuberculous cavity. It is also impossible to detect minute aneurysms of the pulmonary arterioles when situated, as they occasionally are, in the mucous membrane of the bronchi, except by the character of the hemorrhage. When the blood is from the mucous membrane it is usually streaked with mucoid expectoration. When the blood comes from a tuberculous cavity it is generally bright red in color, more or less frothy, coagulates slowly, and may be mixed with mucus and other constituents of the cavity. Aneurysmal hemorrhages of this character generally come on suddenly during the progress of tuberculosis; frequently at night after the patient has been sleeping three or four hours; occasionally upon his rising in the morning or attempting any unusual exertion. One of these aneurysmal dilatations often ruptures after a severe paroxysm of coughing. In quantity the blood may amount to a tablespoonful or it may be as much as a teacupful or even a pint. This hemorrhage, its origin, course, and treatment will be discussed in the section on Hæmoptysis. It merely remains to call attention once more to the fact that pulmonary aneurysms may exist within the pericardium, without the pericardium, or in the main pulmonary artery, and that smaller aneurysms occur in the mucous and submucous tissues of the bronchi, but most frequently at the circumference of tuberculous cavities and occasionally in the cavity itself where the artery stretches across, not having undergone degeneration.

For a consideration of the *treatment* of pulmonary hemorrhage the reader is referred to the section on this subject below.

Hyperæmia of the Lungs.

Definition.—A hyperæmia or congestion of the mucous membrane and parenchymatous tissue of the lungs.

Etiology.

Hyperæmia of the lungs occurs under two forms, viz., *active* and *passive* or mechanical.

Active pulmonary hyperœmia is frequently described as a distinct pathological condition of the lungs, and no doubt it does occur as such without going into a pneumonic condition. Of course we recognize that the first stage of pneumonia or even of a bronchitis is one of hyperæmia, but it may possibly be limited to hyperæmia alone in some cases, without extending to inflammation with exudation. Active pulmonary hyperæmia occurs from exposure to cold and sudden changes of temperature. The writer has frequently noticed pulmonary hyperæmia in persons occupied in preserving meat in cold storage rooms and in artificial-ice factories, where considerable muscular exertion in alternate hot and cold air is necessary. Hyperæmia also occurs as the result of inhaling hot air, as observed in firemen on steamers and in factories. The inhalation of irritating gases in chemical works will also produce hyperæmia of the lungs. Hyperæmia precedes most affections of the lungs such as bronchitis, consolidation, pleuritic effusion, pulmonary collapse, acute tuberculosis, etc. It also occurs in violent fits of coughing, and from alterations in atmospheric pressure, as seen in deep-sea divers, caisson workers, and miners. It accompanies morbid growths, pneumonic processes, etc., and follows collateral fluxion in other parts of the lung, and conditions which disturb the physiological relations normally existing between the air in the lungs and the pulmonary circulation. In a number of cases of poisoning by illuminating-gas which have come under the writer's notice, there was very extensive pulmonary hyperæmia. From many causes there may be an increase in the amount of blood going to the lungs, as when there is an increase of pressure in the arterial system from greater force of the heart's contraction or when narrowing of the arterial circulation elsewhere throws an additional quantity of blood on the pulmonary circulation; this occurs from undue exercise, as in athletic sports, football playing, rowing, running, jumping, etc. Hyperæmia of the lungs will occasionally be met with in bicycle riders, especially if they have been taking long trips or racing. Undue exercise of the physical apparatus in speaking or singing will at times produce congestion of the lungs. The

ingestion of cold drinks such as iced lemonade or the eating of ice cream when the body is warm and perspiring, will sometimes induce extreme congestion of the lungs. Plunging into cold water when the body is warm will drive the blood into the interior organs and induce congestion, which is frequently of a serious nature. In pneumonia, atelectasis, and obstruction of some of the branches of the pulmonary artery, other parts of the lungs will become hyperæmic in consequence of the determination of a greater quantity of blood to them.

Passive or mechanical hyperæmia is generally referable to some heart lesion in which there is considerable interference with the passage of the blood from the lungs to the left cavities. A dilated or weak heart will also produce the condition. In low-grade or long-continued fevers such as typhoid, in which there is cardiac and circulatory weakness compelling the long maintenance of the recumbent position, pulmonary congestion is usual. This, on account of the influence of gravitation, is generally found in the posterior portions of the lower lobes of the lungs and it is then called *hypostatic congestion*. Any tumor or aneurysm which impedes the circulation in the lungs, such, for example, as abdominal tumors or ascites pressing the diaphragm upwards and thus interfering with pulmonary circulation, will produce passive hyperæmia of the lungs. Chloroform or ether narcosis is a frequent cause of pulmonary hyperæmia, and lethal doses of opium or morphine will produce it. Cerebral injuries or lesions often cause pulmonary congestion. Passive congestion of the lungs is produced as a rule by causes interfering with the return of blood from them. Mitral stenosis and insufficiency, aortic stenosis and insufficiency, and obstructive lesions maintaining venous stasis are typical examples of some of the causes of congestion. A weak heart will produce the same results as valvular insufficiency. Cirrhosis of the liver with chronic congestion of the kidneys, stomach, and spleen will in turn produce passive congestion of the lungs. General arteriosclerosis, as found in Bright's disease, will also cause passive congestion of the lungs. Hypostatic congestion, on the other hand, usually results from adynamia. In cases of chronic and long-standing diseases in which the patient is obliged to observe the dorsal position for any length of time, and in old people confined to bed by an injury or some other affection, hypostatic congestion of the lung should always be considered and prevented if possible.

Pathology.

Whereas active hyperæmia or congestion of the lungs depends upon the dilatation of the arterioles, the passive variety is due to

the dilatation of the venules. In either case the result is a greater volume of blood supply to the lungs, causing swelling of the tissues and mucous membrane and leaving less room for the interchange of oxygen from the air and carbonic-acid gas from the blood. The lung tissue is more or less deep-red, bluish, purple, livid, or blackish-red in color, depending upon the amount of congestion. Usually both lungs are implicated and generally at their bases. Occasionally only one lung or a portion of one lung is affected, depending upon the etiological factors involved. Hyperæmic lung tissue floats on water, unless in the very chronic form from valvular disease of the heart, when the pathology resembles that of consolidation, or *splenification* as it is termed; in such cases the lung tissue does not float on water, as the air-cells are practically filled with serum and mucus. Hypostatic congestion frequently passes into hypostatic pneumonia as seen in typhoid and the continuous fevers. The lumen of the minute bronchi is narrowed and less air enters the lungs. If the hyperæmia subsides the blood-vessels and tissues resume their normal physiological condition. Feebleness of circulation induces passive congestion by the retention of too much blood in the lungs, not because the blood supply is greater than normal, but because the amount returned to the heart is less than in a normal condition of the circulation. In congestion of the lungs the parenchyma, as before mentioned, is of a dark-red or bluish color. On section it is found to contain more fluid in the blood-vessels, more fluid in the interstices, and there is more injection of the bronchi than is found in health. Besides this, the interstitial connective tissue is more or less distended with serum, the condition bordering on œdema, and the capillaries are so swollen as to compress the alveoli, in many cases almost if not quite obliterating the air cells. Throughout the lung tissue numerous minute extravasations of blood will be found from rupture of small capillaries.

Symptoms and Signs.

Pulmonary hyperæmia is characterized by more or less dyspnœa and tightness across the chest as a consequence of the swelling of the bronchial mucous membrane and the obstruction to the entrance of air. There is seldom much pain, as actual inflammation and exudation of inflammatory products have not taken place. There may be considerable cough and some watery and mucous expectoration present. At times the hyperæmia is so intense as to cause hæmoptysis, which occurs in both the active and passive forms. Should œdema result from the hyperæmia the expectoration will be watery; and should a bronchial hemorrhage induce inflammation the pneumonic

process is the result. The respiratory movements are at first increased in frequency as less air is admitted to the lungs. When extensive congestion supervenes the respiratory movements are diminished. The percussion note is at first clear or even tympanitic; but when congestion is prolonged or there has been a hemorrhage or there are pneumonic spots or splenification, localized dulness will be elicited. The respiratory sounds are usually weak and harsh; at times bronchial in character with moist fine râles. Sudden complete congestion of both lungs may cause death in a few hours. In other cases the oppression varies from slight discomfort to extreme dyspnœa. The face is flushed or cyanotic, depending upon the severity of the congestion. The pulse is at first full and strong, and there is violent beating of the carotids, with considerable injection and brilliancy of the eyes. The congestion is so severe at times as to set up complete œdema of the lungs, which is a very formidable condition. There is usually considerable headache and throbbing in the temples, considerable cough with little expectoration at first, but after a few hours frothy liquid sputum will be expelled in considerable quantities. The sputum is frequently tinged with blood from the capillary hemorrhages in the bronchial mucous membrane. The vesicular murmur is generally more or less absent and in its place subcrepitant and mucous râles are heard over the congested portion of the lungs. In extreme cases the alveoli are filled with fluid and the breath sounds approach bronchophony in character. When the air cells are so filled with serum or when the congestion is so great as to compress them and prevent the entrance of air, oxygen cannot reach the blood, hence there is an accumulation of carbonic-acid gas. The blood is filled with hæmoglobin charged with carbonic-acid gas, instead of oxyhæmoglobin charged with oxygen. This explains why the lips are blue, why there is headache, and why the face is livid. When the congestion is severe, the pulse, which was at first strong and bounding, will soon become small, thready, and intermittent. The surface of the body will be covered with cold, clammy sweat, the fingers will become bluish from the accumulation in the blood of carbonic-acid gas. There will be extreme restlessness and shallow breathing; the respirations will frequently reach 40, 50, and 60 per minute, and the restlessness will be followed by drowsiness and finally coma. As the stupor increases there will be less effort to cough and inability to expel the accumulated secretion in the bronchi. At the same time the breathing will become more difficult as the bronchial tubes fill and become occluded, until in fatal cases both lungs become entirely filled. The termination of chronic heart disease and chronic Bright's disease is usually by extreme congestion

of the lungs with œdema. It should be remembered that a congested lung is very prone to take on inflammatory action, and that if the congestion be not relieved pneumonia is very apt to supervene.

Diagnosis.

The etiological factors productive of hyperæmia must be well considered. There is usually little if any fever. In the active variety the cause is generally apparent; in the passive form the cause must be searched for. In general, the harsh breathing, without crepitation or dulness, slight mucous or watery expectoration, dyspnœa and tightness of chest, and the etiological factors will be sufficient to establish the diagnosis. From the absence of chill and fever it will at once be seen that the condition is not one of inflammation. From the shallow, rapid breathing and dyspnœa it will be noticed that the lungs are affected; from the absence of vesicular murmur, with bronchial breathing, bronchial râles, and at times vesicular crepitation, it will not be difficult to establish the fact that the lungs are engorged. In the acute variety there is generally some explanation in the way of overexertion, sudden chilling of the body, etc., and in the passive form of the disease the heart, kidneys, liver, and other organs will be found more or less responsible for the condition of the lungs.

Prognosis.

In the acute hyperæmia of overexertion, exposure to irritating gases or to hot or cold air, of collateral fluxion, etc., the prognosis is good if the cause can be removed and the circulation can be reestablished; but in the passive form from heart disease, pneumonia, tumors, pleuritic effusion, tuberculosis, hemorrhages, Bright's disease, etc., where the cause cannot be removed the prognosis is much more serious. When the hyperæmia goes on to hypostatic or acute pneumonia, œdema, and the formation of abscesses, the prognosis is necessarily grave, not only because of the hyperæmia but also on account of the etiology and of the complications.

Treatment.

The first indication is to remove the cause, if possible. In active hyperæmia of the lungs a hot bath of 105° to 110° F. for five or ten minutes will often prove of great benefit. If the hot general bath is not thought advisable or practicable, local applications of turpentine stupes, sinapisms, or poultices, dry cupping or even blisters on the chest, front and back, will often afford relief by directing the

blood to the surface. Not that there is any direct communication between the blood supply of the surface of the chest and that of the lungs, but by dilating the cutaneous vessels we afford the circulation an opportunity to re-establish its normal distribution. In severe active congestion of the lungs and in the passive variety, when the patient is of a plethoric habit and strong, phlebotomy is of undoubted benefit. When the pulse is full and bounding and active hyperæmia of the lungs exists, if bleeding be deemed inadvisable, half a drop to a drop of the tincture of aconite may be administered every fifteen minutes for the first hour or two at the onset of the disease. This will relax the capillary system throughout the body and determine the blood away from the congested organs. The tincture of veratrum viride, in five to ten minim doses, has a similar effect to that of aconite and may be employed under the same conditions. The next indication in active hyperæmia is to produce diaphoresis. For this purpose the tincture of jaborandi in half-drachm or drachm doses every hour for two or three hours, or pilocarpine hypodermically in doses of from one-tenth to one-fifth of a grain every fifteen minutes for two or three doses, will prove very satisfactory. The induction of a profuse perspiration may abort hyperæmia and will often check it to a considerable extent. In patients of a rheumatic diathesis the sodium and lithium salicylates, in five to ten grain doses every hour for four hours, will assist in quieting the circulation and in reducing the pulmonary blood supply. Other diaphoretics, such as ipecacuanha, tartar emetic, sulphur, camphor, spirit of nitrous ether, ammonium acetate and citrate, potassium acetate and citrate, mezereon, etc., will prove of value. Active diuresis is also indicated in engorgement of the lungs. This may be rapidly induced by remedies which dilate the renal arteries, but it would be unwise to give those diuretics, such as digitalis or squill, which raise the general blood pressure, for we desire general arterial relaxation as much as diuresis. But remedies which stimulate the kidneys, as sodium or potassium nitrite, nitrate, acetate, citrate, etc., caffeine citrate, juniper, scoparius, buchu, and lobelia, will prove of considerable benefit. The bowels must also be called upon to assist in eliminating the watery portion of the blood. Salines are useful for this purpose; one of the most active of them is magnesium sulphate, a saturated solution in peppermint water, of which a teaspoonful may be given every fifteen minutes until free catharsis occurs. The sulphates of sodium and potassium or the tartrate of potassium and sodium are also useful. Ten or twenty grains of calomel, followed by a saline, is an excellent cathartic.

In chronic hyperæmia the cause of the congestion should be treated. Use strychnine and digitalis in mitral disease, evacuate a pleuritic

effusion or an ascitic abdomen. In typhoid and continued fevers frequent changing of the patient from side to side is of importance. Frequent deep or forced inspirations are to be recommended. Stimulating expectorants may assist in preventing œdema or inflammation, and inhalations of warm air or steam with ammonium chloride are beneficial.

Pulmonary Hemorrhage.

Pulmonary hemorrhage is a subject of grave importance. It comprises hemorrhage from the respiratory tract and hemorrhage from and *into* the lung substance itself. For convenience of description pulmonary hemorrhage may be divided into—(1) Diffuse or true pulmonary; (2) Nodular or circumscribed; (3) Interlobular, and (4) Petechial.

Diffuse or True Pulmonary Hemorrhage—Hæmoptysis.

Definition.—Hemorrhage from the air passages or lungs, spitting of blood. Tracheal or bronchial hemorrhage will produce hæmoptysis; so will *diffuse* pulmonary hemorrhage excepting that form which ruptures into the pleural cavity producing pneumothorax. But there are several forms of pulmonary hemorrhage which do not produce hæmoptysis which will be mentioned later on. The writer would impress upon the reader the fact that *pulmonary hemorrhage* does not necessarily mean hæmoptysis nor any external visible sign of blood, and that *hæmoptysis* does not necessarily mean true pulmonary hemorrhage.

Etiology.

Hæmoptysis may occur from any part of the air passages, such as the nose, throat, trachea, bronchi, or lungs. Any hyperæmia, inflammation, ulceration, or injury of the respiratory tract may produce hemorrhage or spitting of blood. One of the most frequent causes of hæmoptysis is phthisis. In the early period of the disease small hemorrhages may occur from congestion of the tuberculous site. In the later stages large hemorrhages may result from the rupture of a blood-vessel weakened by tuberculous ulceration of the tissue about it, or from a small aneurysmal dilatation of the end of an artery in a cavity. The hemorrhage in phthisis is more or less characteristic of the disease. In many cases occurring in California hemorrhage is one of the earliest signs. For several hours daily small pellets of blood are expectorated with little or no other sputum. The blood is generally bright red and frothy, gradually becoming darker and

darker as the slight hemorrhages become less frequent; in five or six days or earlier the bleeding ceases entirely. In the later stages of phthisis the muco-purulent sputum is often streaked or stained with blood which comes generally from the bronchial mucous membrane. Profuse and alarming hemorrhages occur from the rupture of aneurysmal bulbs in a cavity or from the rupture of an ulcerated vessel; it may be from two or three ounces up to a pint or more in quantity. This constitutes the most frequent form of true pulmonary hemorrhage.

Traumatism such as gunshot wounds of the lung or other injuries will cause hæmoptysis and pulmonary hemorrhage. Rupture of an aneurysm into a bronchial tube will produce hemorrhage usually of an alarming nature. Gangrene of the lungs may likewise be a cause. Primary irritative causes, such as the inhalation of chlorine gas, dust, etc., may produce hemorrhage by inducing congestion and rupture of bronchial capillaries. Prolonged exertion of any kind, such as mechanical straining, lifting, singing, great mental excitement, etc., frequently produces hæmoptysis. Inherited abnormal weakness of the blood-vessels as found in strumous children is an important factor in the production of hemorrhage. Hæmophilics, the so-called "bleeders," are predisposed to pulmonary hemorrhage on the slightest provocation. Congestion of the lungs from any cause, as in valvular disease of the heart, is frequently responsible for hæmoptysis. Vicarious hemorrhage occasionally occurs from the bronchial mucous membranes in suppressed menstruation. Flint says he has known vicarious menstruation to occur from the lungs at regular intervals for four years after the suspension of the menses. In croupous pneumonia, fibrinous bronchitis, emphysema, fibroid phthisis, syphilitic pneumonia, and œdema of the lungs, the expectoration may be streaked with blood, although it is generally more or less intimately mixed with the sputum. Carcinoma, syphilis, and other ulcerations of the bronchi and lungs usually produce hæmoptysis. In fibrinous bronchitis there are often early attacks of hæmoptysis. Chronic naso-pharyngeal catarrh is sometimes attended with spitting of blood, as is also the removal of diphtheritic membrane. In emphysema and chronic bronchitis blood is occasionally coughed up. Sir Andrew Clark mentions a form of hæmoptysis which he calls "arthritic," occurring in persons suffering from the arthritic diathesis. Hæmoptysis may also occur from the lodgment of an embolus in one of the branches of the pulmonary arteries, especially if the embolus be septic, or from nodular pulmonary hemorrhage. In the course of scurvy, purpura, pyæmia, and malignant fevers the sputum may become sanguineous. Diminished atmospheric pressure will often cause hæmoptysis. The writer has fre-

quently observed this in strong men when climbing mountains at an altitude of over ten thousand feet.

Many women hysterically inclined, suffering more or less with anæmia, some form of uterine irritation, and many fancied ailments, not infrequently cough up a little blood. This naturally alarms the friends exceedingly, but it seems to be of very little importance and depends mainly on some local congestion or unusual exertion. Pregnant women and nursing mothers sometimes have hæmoptysis. In chronic valvular diseases of the heart, aortic or mitral, when there is considerable hyperæmia or chronic congestion of the lungs, slight bleeding from the bronchial capillaries of the mucous membrane is usual. The hæmoptysis may be quite considerable at times, especially after a full meal, after a severe paroxysm of coughing, or after some unusual exertion. One or more of the larger capillaries may give way and the blood pours into the bronchial tubes from which it is expelled by coughing. In passive congestion from heart disease the blood is generally of a dark color. Alarming hæmoptysis occurs from the rupture by aneurysmal dilatations of the branches of the pulmonary arteries within the lungs. Aneurysms of the larger blood-vessels, as of the arch of the aorta, often erode the trachea or one of the main bronchi and rupture into these tubes by large or small openings. In such cases the pulmonary hemorrhage is usually at first small, the blood being bright red, but it is more or less continuous for a few minutes, or a few hours, in which time a considerable quantity may be coughed up. The hemorrhage becomes more alarming in the course of a few hours or days, and at any time on the slightest exertion the patient may die from the sudden loss of a great quantity of blood and from choking up of the bronchi. In these aneurysmal hemorrhages the blood frequently is sucked into the smaller bronchi and thence into the air cells, setting up a peculiar form of pneumonia having subcrepitation and crepitation from the onset. This, of course, will be recognized as hæmoptysis and subsequent pneumonia from the introduction of blood into the air cells.

Pathology.

The pathology of hæmoptysis consists in the rupture of a vein or artery, pulmonary or bronchial, from some of the many causes enumerated in the section on etiology. The blood may come from a highly congested mucous membrane with a capillary rupture or from inflamed or ulcerated tissues in or about a blood-vessel. As the most frequent form of hæmoptysis is found in phthisis it is easy to understand how the eroded and ulcerated tissues will weaken a

vessel and cause its rupture. The pathology of hæmoptysis is of course the rupture of a blood-vessel. The causes leading to this rupture may be simple congestion from chronic valvular disease of the heart or congestion from any of the many causes, such as hyperæmia from a cold, bronchitis, etc. In hæmophilia any unusual exertion or the slightest congestion of the mucous membrane of the lungs may cause hemorrhage. In these cases there would seem to be a special predisposition to hemorrhage either in the blood-vessels themselves or in the blood, or both. The blood-vessels of sufferers from hæmophilia rupture on slight provocation, probably from the want of elastic or contractile material in their walls, which show no disposition to contract or retract and thereby stop the hemorrhage. The blood itself seems to be more liquid, and to pass more readily through the vessels walls. In vicarious menstruation the same explanation must be made, namely, that certain circumscribed spots of the mucous membrane become congested. Capillaries rupture and bleeding occurs. This, as we know, may take place from the rectum and bowels, from the stomach, from the nose, eyes, throat, or lungs; in fact any mucous membrane, even the bladder, may be the seat of vicarious menstruation. When the blood comes from the lungs for any length of time it is of graver import, as some of it may enter the air cells and set up lobular pneumonia, or it may form a favorable nidus for the lodgment of tubercle bacilli.

Symptoms.

Hæmoptysis is easily diagnosed, and from the symptoms and physical signs it is usually not very difficult to ascertain the cause of the hemorrhage or the anatomical location of the bleeding vessel. Blood from the respiratory organs is generally expelled by coughing, and unless it is very copious, it is usually of a florid frothy character. If the blood comes from the nose, careful inspection will usually determine the fact. A pledget of cotton or a probe passed into each nostril and carried well back to the posterior nares is almost certain to determine whether the hemorrhage is nasal or not. The same examination with an illuminated mirror will determine whether the blood comes from any part of the pharynx or larynx. To ascertain whether the blood comes from the stomach or from the respiratory organs it is necessary to remember that blood from the stomach in hæmatemesis is generally dark or even quite brown in color because of the longer stagnation and the effect upon it of the secretions of the stomach; it is often mixed with food, or it may be like coffee grounds or clotted and it generally has an *acid reaction*. Blood from

the lungs, on the other hand, has a bright-red color and is often frothy or mixed with mucus or pus. It is seldom clotted and is generally of an *alkaline* reaction. Still, it must be remembered that—

1. In hemorrhages from the respiratory tract a considerable amount of blood may be swallowed and subsequently vomited; 2. When a hemorrhage from the lungs is profuse the blood may pour from the mouth without any attempt at coughing; 3. If blood is derived from an apoplectic effusion in the lungs it may be almost black in color; 4. In hæmatemesis and in epistaxis blood may sometimes find its way into the bronchi and be coughed up; 5. In an aneurysm of the arch of the aorta or of one of the branches of the pulmonary artery which has eroded into the trachea or a large bronchus there is usually a profuse and often fatal hemorrhage. At times the eroded point is quite small and the patient coughs up a little blood from time to time, while some of it escapes into the air cells and sets up a pneumonia.

As a rule—

a. When hæmoptysis is slight it is generally from the bronchial mucous membrane, as in bronchitis, congestion, or from a pneumonia or a pulmonary apoplexy; *b*. When it is profuse the blood is generally from the rupture of an eroded blood-vessel in a phthisical cavity, or from the rupture of an aortic or other aneurysm, or from a syphilitic or cancerous ulceration.

When there is hemorrhage with tickling in the throat, coughing, dulness on percussion over a limited area, and crepitation, it is pretty sure that the blood is from the lung. On the other hand, when there is nausea, faintness, dizziness, and vomiting of dark-colored blood of an *acid* reaction with no pulmonary symptoms, it is pretty sure that the blood comes from the stomach. In hæmatemesis there is usually also melæna following.

Prognosis.

The termination of hæmoptysis depends entirely on the cause of the hemorrhage. In the rupture of an aneurysm or the breaking of a vessel in a phthisical cavity the violent outburst may cause the death of the patient suddenly from syncope or from the exclusion of air from the bronchi in consequence of the large quantity of blood filling the tubes. Hæmoptysis is generally slight and rarely fatal, although the conditions which induce it may lead to grave results.

Treatment.

In most cases the hemorrhage must be treated by addressing remedies to the causative disease. In the small hemorrhages oc-

curring in the course of chronic nose and throat troubles, bronchitis, œdema, cardiac congestion of the bronchi, and early stages of tuberculosis, no special treatment is necessary. When, however, the hemorrhage is profuse and threatens to become dangerous, active measures must be promptly employed. The patient should lie down and keep perfectly still; the circulation should be quieted, and the patient's anxiety should be relieved; no exertion of any nature should be permitted, and even speaking should be prohibited. The apartment should be cool, the clothing light. Ice-bags may be applied to the chest, nose, and back of the neck. Ice or very cold acid drinks may be employed with advantage. Astringents such as lead, gallic and tannic acids, and ergot may be administered. Ergotin, gr. ij.–v. hypodermically, has proven of great value in the writer's experience. Atropine, gr. $\frac{1}{100}$ hypodermically, has proven invaluable in stopping the hæmoptysis of phthisis. Ipecacuanha in 5 or 10 grain doses to produce emesis has been recommended in hæmoptysis. Turpentine in doses of 30 to 60 minims on sugar or in an emulsion every few hours will sometimes stop the bleeding. Alum in 10-grain doses is serviceable. Oil of erigeron, hamamelis, digitalis, ferric acetate, matico, krameria, infusion of arnica, rhatany, copper sulphate, and aconite have all proven of value. Iron spray and alum spray have been employed with good results. Teaspoonful doses of sodium chloride, dry or in water, will at times arrest hæmoptysis. The tincture of the chloride of iron, in drachm-doses in ice-water every fifteen minutes, is of value. Sulphuric acid, vinegar, or citric acid, and iced lemonades are of value. Full doses of ergot and laudanum, ℥ xxx. of each every hour, have proven of great benefit in the writer's hands.

Circumscribed or Nodular Hemorrhage.

Definition.—Circumscribed or nodular pulmonary hemorrhages occur from the rupture of the capillaries or of the small pulmonary arterioles in the lungs. The blood collects *within* and at times *outside* of the alveoli as well as in the minute bronchi, without any laceration of pulmonary tissue. There may or there may not be hæmoptysis. This form of pulmonary hemorrhage is termed *hemorrhagic infarction* or *pulmonary apoplexy.*

Etiology.

Circumscribed pulmonary hemorrhage is most frequently due to hemorrhagic infarctions. An embolus from the right cavities of the heart, as in malignant endocarditis, from the systemic veins as

in phlebitis, or from some pyæmic infection, may lodge in one of the smaller branches of the pulmonary artery, and a circumscribed hemorrhage is the result. This form of pulmonary hemorrhage is also caused by acute lobar pneumonia; more frequently by lobular pneumonia. Not infrequently it is caused by pyæmic infection, advanced Bright's disease, or thrombosis of the pulmonary artery, in cases of debility or other causes, leading to impoverishment of the blood, when coagulation is prone to occur.

Pathology.

When a small blood-vessel is stopped by an embolus, thrombus, or a plug composed of septic bacteria, hemorrhage usually results. The blood escapes into the interalveolar texture of the lungs and air cells. Hæmoptysis may be present and it may not. When blood lodges in the interalveolar tissues from an infarction or nodular hemorrhage it forms an extravasation which is known as *pulmonary apoplexy*. The clot may vary in size from that of a pulmonary lobule up to that of a small orange or even larger. The hemorrhagic infarct and consequent extravasation occur most frequently in the outer borders of the lower lobes of the lungs. The infarct affects one or more lobules, and the clot is more or less pyriform in shape with the base toward the pleural surface. In color the tissues and clot are dark or reddish-black and contain very little if any air. As the blood clot contracts frothy serum oozes out. These apoplectic clots undergo decolorization in the same way as does an effusion of blood into the subcutaneous tissues. If small, complete absorption may occur, leaving behind only brownish granular pigment. Should the embolus causing the stoppage of the vessel and consequent apoplexy be septic in character, as occurs in malignant endocarditis and pyæmic infection, the clot softens, breaks down, and induces inflammation, pneumonic spots, and abscesses of a very grave nature. The pus from these abscesses may escape into a bronchus and be expectorated, but not infrequently the abscess opens into the pleural cavity, setting up septic pleurisy, pneumothorax, and empyema. Pulmonary infarcts when near the surface of the lung are wedge-shaped, as stated above, but when they occur deep in the substance of the lung they are usually larger and composed of dark-colored blood with surrounding pneumonic inflammation. Unless degeneration occurs, as in the septic variety, the infarct and apoplectic clot become hard and firm, airless and granular. When absorption is effected a dark-colored pigmented spot remains, with more or less consolidated lung tissue to mark the spot. At times the clot undergoes caseous or calcareous

degeneration and may become encapsulated. In rare cases a pulmonary extravasation may lead to a gangrenous spot, which decomposes and is either expelled by coughing with subsequent resolution of the tissues, or it ruptures into the pleural cavity, setting up pneumothorax or empyema.

Symptoms.

These are similar to those of hæmoptysis. There may be hæmoptysis. There is usually dyspnœa, with tightness and oppression across the chest, and more or less cough with or without much expectoration according to the location of the apoplexy. Pulmonary œdema and lobular pneumonia not infrequently supervene.

Diagnosis.—A circumscribed or nodular pulmonary hemorrhage, an infarct, and an apoplexy are diagnosed by their etiological factors such as valvular disease, phlebitis, pyæmic processes, and hæmoptysis if present, and local and systemic symptoms such as dyspnœa, pneumonic inflammation, pyrexia, cough, etc. The presence of Bright's disease is an important factor in the absence of cardiac or obvious pulmonary complications.

Treatment.

This is the same as that of hæmoptysis. The complications arising in the pulmonary tissues, as well as the cardiac, renal, and pyæmic diseases which produced the hemorrhage, must also be carefully considered.

Interlobular and Petechial Hemorrhages.

These occur in the course of constitutional disorders such as pyæmia, puerperal fever, diphtheria, purpura, typhus, small-pox, and malignant septic fevers. The etiology, pathology, and treatment are essentially those of the disease producing them. When pulmonary congestion, inflammation, pneumonia, abscesses, or gangrene supervene in the course of any of these severe constitutional diseases the case is almost hopeless, and the practitioner cannot be too much on his guard in reference to the prognosis. The lungs and sputum should be examined frequently, the possible complications carefully watched, and such remedial measures instituted as the exigency of the case requires from the symptomatic indications.

Œdema of the Lungs.

Definition.—The transudation of serous fluid into the air cells and pulmonary areolar tissue.

Etiology.

Prominent among the etiological factors in the causation of œdema of the lungs are valvular disease of the heart and acute and chronic Bright's disease. Any disturbance of the circulation may, however, also cause œdema. In this manner tumors and aneurysms may press on the pulmonary vessels and produce localized œdema. In elderly people with failing circulation, œdema is not infrequent. In long-continued fevers, such as enteric, there is frequently an hypostatic congestion, surrounded by œdema, which may take on inflammatory action such as acute pneumonia. In extensive pneumonitis or pleuritis of one side, the healthy lung may become œdematous and thus contribute largely to a fatal termination. Other causes of œdema are: Overexertion of the vocal organs in speaking and singing; the inhalation of cold or hot air, or the sudden transition from one extreme of temperature to the other; the inhalation of irritating gases or vapors; ingestion of ices and cold drinks when the body is very warm; the sudden chilling of the surface of the body when warm, as by plunging into a cold bath. Œdema is frequently seen in emphysema of the lungs as a result of the congestion and impeded circulation. Congenital heart affections, which are usually on the right side, are very liable to produce chronic hyperæmia and œdema from the imperfect circulation between the heart and lungs. Fibrous hepatitis also interferes with the normal pulmonary circulation and may cause œdema. Œdema of the lungs is also seen in suppurative hepatitis, Hodgkin's disease, amyloid liver, puerperal eclampsia, polyuria, and acute rheumatism. Pericarditis, from its interference with the heart's action, is also productive of this condition. Sero-fibrinous and purulent pleurisy, by producing adhesions may set up peripheral pulmonary congestion and consequent œdema, which may prove serious. Leukæmia and anæmia are important factors in the production of œdema, and many cases of primary or secondary anæmia as well as the anæmia known as chlorosis or "green sickness" show great proneness to pulmonary œdema on the slightest provocation. This is not at all strange when we consider the hydræmic state of the blood, the paucity of red blood corpuscles, and the poorly nourished blood-vessels and tissues generally. Anæmic patients and chlorotic girls are easily bruised; the lightest pinch or blow produces considerable ecchymosis; the

blood-vessels are not firm and elastic and the blood has a lighter specific gravity, hence serous transudation occurs easily; œdema in an anæmia is often seen about the ankles and eyelids; the bronchial mucous membrane may become œdematous on the slightest over-exertion; and by reason of the weakened condition of the heart and circulation and impoverishment of the blood serious pulmonary œdema is often set up. This may be explained in one of two ways. First, there can be little doubt as to the abnormal permeability of the vascular walls generally in anæmia, and any extra exertion calls for greater blood supply, and as a consequence there is greater distention of the already weakened blood-vessels with the thin watery blood; hence the obstruction to the circulation effected by the exertion would seem to be sufficient to cause transudation of serum and œdema of the lungs. The second cause of pulmonary œdema occurring in the course of anæmia may be ascribed to passive congestion of the lungs caused by cardiac debility. The poorly nourished heart sends the blood with diminishing force into the lungs and it is returned to the heart with little force. The normal physiological relation has been destroyed and a slow chronic passive congestion of the lungs ensues. Upon this passive congestion œdema of the lungs readily supervenes, and it is always more or less serious on account of the causative conditions present in the impoverished blood and debilitated heart.

Another etiological factor in the production of œdema of the lungs is acute ascending spinal paralysis. In the course of this usually rapid disease an œdema may appear at first in the lower extremities. In a few days great difficulty may be experienced in drawing a breath and on examination extensive œdema of both lungs will be found. The *modus operandi* would seem to depend upon motor paralysis. The lungs become hyperæmic and transudation of serum rapidly ensues. Owing to the seriousness of Landry's paralysis—the cause of the œdema—many cases go from bad to worse and die in a few days unless sufficient stimulation can be exerted to re-establish respiration and the pulmonic circulation. Diabetes at times produces a cutaneous and submucous œdema, which if it comes on in the later stages of the disease may prove very rapidly fatal. Cancer of the stomach or other organs and pernicious cachectic conditions generally are occasionally the remote cause of œdema of the lungs. The blood becomes impoverished and filled with foreign bodies, such as *microcytes* and *poikilocytes*, which impair the nutrition of the vascular walls and cause hydræmia and cardiac weakness. Whenever the heart is weak and the blood is in an impoverished condition, œdema of the lungs is not of infrequent occurrence; in fact, after any unusual exertion such as getting out of bed or strain-

ing at stool, many of these cases terminate in œdema of the lungs and death.

Acute Angioneurotic Œdema.

In the œdema depending upon vasomotor or trophic disturbances the acute angioneurotic variety is prominent. The peculiarity of this form of œdematous swelling is that it may appear suddenly in various parts of the body without any warning. The patient's health is usually perfectly good or there may be some little gastric disturbance with attacks of gastralgia and vomiting, when suddenly the œdema affects the pharynx, larynx, or bronchi, producing alarming and often dangerous symptoms. The attacks may disappear in a few hours and the patient then returns to his normal condition, with the exception that he is a little weak and exhausted from the effect of the pseudo-paralysis of the vasomotor system in the mucous membranes of the throat and lungs. This acute angioneurotic œdema is a malady which seems to be closely allied to urticaria and erythema. The swelling, instead of occurring on the skin as in the latter diseases, takes place in the mucous membranes of the throat and bronchi and in the lungs. By reason of the greater distensibility and greater supply of dilatable blood-vessels in the mucous membrane a greater amount of swelling occurs. At times this œdema is so severe that breathing is seriously interfered with. The patient becomes cyanotic, the heart's action is at first strong and labored but it soon becomes weak, and the right cavities are prone to undergo acute dilatation from the great obstruction to the circulation in the lungs.

The exact cause of this form of œdema would seem to reside in the blood itself. Some irritating or unoxidizable product of digestion sets up a vasomotor paralysis in different parts of the body, and the consequent dilatation of the blood-vessels and transudation of serum into the surrounding cellular tissues is the explanation of the sudden attack of œdema.

Pulmonary œdema may also be due to congestion and inflammation of the respiratory organs or to cardiac disease or other affections involving obstruction to the venous circulation; it may also arise in the course of renal disease, blood-glandular diseases, and nervous affection.

Pathology.

Œdema occurs from venous stasis, hyperæmia, and inflammation as well as from altered conditions of the blood and blood-vessels. Cohnheim's theory of œdema was that the endothelium of the vessels was injured by the stasis, hyperæmia, or inflammation, thereby al-

lowing transudation of serum into the surrounding intercellular tissues.

Landerer advanced the theory that the "loss of elasticity and diminished squeezing-out of lymph from the tissues by their being relaxed" play an important, perhaps a chief, part in causing œdema (Vierordt). When a ligature is tightly bound about a limb, œdema follows. This is undoubtedly due to the obstruction to the circulation and increased pressure within the vessels. In hyperæmia a similar condition obtains in and about the hyperæmic area, and the same holds good in and about an inflamed region. From the obstruction there is also a relaxation of the vessel walls, due to the nervous force being impeded. Under such conditions œdema forms. The œdema of children and anæmic girls, and that occurring in Bright's disease would seem to depend upon the condition of the blood, as well as upon the relaxant influence of malnutrition upon the vessels and tissues. Œdema of the lungs is a frequent accompaniment of general anasarca from whatever cause. It is most frequently observed in connection with scarlatinal tubulitis and other forms of Bright's disease, and in cardiac valvular complications. Œdema is chiefly observed in the most dependent parts of the lungs or in the vicinity of congestion and inflammation. Acute œdema may come on in a few hours in the course of diseases of the kidneys without apparent cause other than the alteration in the blood and in the blood pressure, or the general arteriosclerosis. An œdematous lung is usually enlarged, heavy, pale, pits on pressure, and on section a large quantity of serous fluid mixed with air bubbles will drain away or may be squeezed out. The lung is rarely solidified, although it always contains much less air than normally. Œdema of the lungs not infrequently results in inflammation and pneumonic consolidation. There is serous infiltration into the interstitial connective tissues and into the alveoli.

Symptoms.

Œdema of the lungs produces quickened labored breathing, rattling, and dyspnœa. All the accessory muscles of respiration are called into play. There is a feeling of tightness or oppression across the chest, and the patient generally sits upright in bed. Pain is ordinarily absent. There is cough with profuse watery, blood-stained, and frothy expectoration. Cyanosis is more or less marked. In simple œdema there is seldom any fever; the temperature may even be subnormal.

Physical Signs.

Inspection.—There are great anxiety of the countenance, rapid and violent action of the heart-beating carotids, hasty and troubled cough with more or less frothy liquid expectoration. As the œdema becomes more pronounced the circulation is more and more impeded, the lips become cyanosed, and the fingers and general surface blue and cold.

Palpation furnishes little of importance.

Percussion.—There is more or less dulness, never complete, on both sides of the chest, most marked at the back over the dependent portions of the lungs.

Auscultation.—The respiratory murmur is feeble or absent. There are, during inspiration, abundant subcrepitant and crepitant râles, resembling those of pneumonia but more liquid. When the air cells are filled the respiration will be tubular, approaching bronchophony.

Diagnosis.

Œdema of the lungs is usually easily made out from the history of the case. In œdema there is no chill nor elevation in temperature; there are abundant moist, watery râles and serous or sero-sanguineous expectoration; both sides are generally involved and the most depending parts of each lung posteriorly are first filled. The crepitation is similar to that found in pneumonia but is more general and more watery; the dyspnœa and cyanosis are greater than in pneumonia and the onset is without chill, fever, or pain.

Prognosis.

This is always serious. Most cases of pneumonia, chronic heart disease, and acute and chronic kidney disease terminate fatally from pulmonary œdema.

Treatment.

The treatment of œdema must depend upon the severity of the case and its cause. In most cases, excepting the very acute variety, the heart, liver, and kidneys will be found seriously affected and the treatment must be directed towards the establishment of the normal physiological functions of these organs. When the heart is weak strychnine hypodermically, in doses of gr. $\frac{1}{60}$ to gr. $\frac{1}{30}$ every fifteen minutes until gr. $\frac{1}{10}$ has been taken, will frequently stimulate the heart sufficiently to re-establish the circulation in the lungs. Spirit of

camphor and aromatic spirit of ammonia in thirty minims of each, compound spirit of ether in one-drachm doses every fifteen to thirty minutes, tincture of musk in drachm doses, and strong black coffee, freshly prepared and administered per enema, are also valuable heart tonics. In many conditions of impeded pulmonary circulation when the heart begins to show symptoms of weakness and *rapidity* of action, fresh infusion of digitalis in half-ounce doses is of great value. Should the immediate action of the drug be desired, digitaline may be given hypodermically in doses of gr. $\frac{1}{100}$ to gr. $\frac{1}{50}$ every hour until the physiological action of the drug is established. In œdema of the lungs occurring in Bright's disease, jaborandi in ʒ ss.–i. doses of the fluid extract, or pilocarpine hypodermically in doses of gr. $\frac{1}{10}$, administered once or twice according to the action of the heart, will generally induce profuse diaphoresis. Magnesium sulphate may be given in half-drachm doses every hour until large watery evacuations take place. Cream-of-tartar water is also a good remedy as it acts on both kidneys and bowels. An enema of four ounces of the simple or compound infusion of senna will produce free movement of the bowels. When the lungs are much engorged and the heart is weak, squill, digitalis, buchu, or the acetate of sodium or of potassium may be of service as diuretics. Pulmonary œdema may often be relieved by the use of senega, serpentaria, Hoffman's anodyne, ammonium iodide, ammonium carbonate, and other stimulating expectorants. Active emesis, obtained by gr. $\frac{1}{10}$ of apomorphine hypodermically, will frequently relieve a water-logged lung; but emetics should be employed with caution, especially if the heart is weak and the circulation is poor. In acute œdema in persons of full habit of body and a strong heart, venesection is sometimes of exceeding benefit. A few ounces of blood taken from the arm in such cases will generally relieve the pulmonary circulation very satisfactorily. Phlebotomy is not advisable in all cases of acute œdema, but in selected cases the result is almost certain to be beneficial. Hot stimulating drinks and hot enemata will often assist materially in establishing a stronger pulmonary circulation, and the application of hot poultices, turpentine stupes, etc., will often relieve the blood pressure in the lungs and so promote absorption of the fluid. The patient should be frequently changed from side to side in order that the most dependent part of the lungs may not suffer from hypostatic congestion and inflammation. The air which the patient breathes should be moist and warm and the best of stimulating and easily absorbable food, such as hot milk, hot beef tea, broths, etc., should be administered every three hours.

PULMONARY EMPHYSEMA.

Definition.—The word emphysema is from the Greek ἐμφυσάω, to inflate. It is the abnormal collection or infiltration of air in the connective tissue of a part, causing swelling and crepitation.

Pulmonary emphysema is divided into (*a*) Interlobular (interstitial), (subpleural), and (*b*) Vesicular (alveolar). In the *interlobular* and *subpleural* varieties the air is extravasated into the subpleural and interlobular cellular spaces, in consequence, usually, of rupture of some of the acini. It is a disease of childhood almost exclusively. The *vesicular* form consists of dilated and ruptured pulmonary alveoli communicating with one another.

Varieties of Vesicular Emphysema (*Alveolar Ecstasis*).—Vesicular emphysema may be divided into several well-marked varieties, viz.:

1. Acute emphysema: (*a*) general; (*b*) local; 2. Chronic hypertrophous or substantive emphysema (the "large-lunged" of Jenner); 3. Chronic compensating, limited or lobular emphysema; 4. Atrophous or senile emphysema (the "small-lunged" of Jenner).

Etiology.

The great etiological factor of pulmonary emphysema is undoubtedly loss of elasticity in the lung tissues.

Among the primary or exciting causes of vesicular emphysema are diseases of the respiratory organs, in which there is interference with the outlet of air; occupations, such as glass-blowing or playing on wind instruments; excessive athletic exercises, habitual straining at stool, etc. I have observed several cases of emphysema in people who are not robust but who have taken to riding the bicycle.

Poorly developed persons with hereditary weakness of the chest walls are predisposed to emphysema. It is not infrequent in California to see the children of those suffering with phthisis, asthma, or emphysema become themselves emphysematous.

Cardiac diseases which produce pulmonary congestion often do so by inducing degenerative changes in the lung which lead to weakness of the air vesicles. A potent factor, which is generally overlooked, is chronic interstitial fibrosis of the alveolar tissue in the lung, similar to cirrhosis of the liver, kidneys, and general arteriosclerosis. The fact that gouty patients are predisposed to emphysema would corroborate this view. The following are the principal theories concerning the production of emphysema:

1. *Inspiratory Theory.*—This theory assumes that the air vesicles

become overdistended during inspiration, in consequence of diminished expiratory force due to the loss of elasticity of the lungs and chest walls. Laennec supported this theory, believing that the air vesicles became distended on inspiration, and that then, in consequence of obstruction in the bronchi and loss of contractile power of the vesicles, emphysema was produced. Dr. C. J. B. Williams held that the mucous secretions formed in bronchitis obstructed *some* of the bronchi, thereby preventing air from entering the terminal vesicles, the air cells of other free bronchi becoming stretched, overdistended, and ruptured. The same reasoning applies to the collapse of a portion of a lung, consolidation, pleuritic adhesions, or obstructions to the movement of air in a part of the respiratory tract from any cause; in which case the air vesicles in unobstructed portions of the lung would become overdistended and produce the disease. This would be called vicarious emphysema.

2. *Expiratory Theory.*—The expiratory theory of emphysema supposes that the less supported portions of the lungs, such as the apices, the anterior margins, and edges, become overdistended upon violent expiratory effort, when there is obstruction from any cause, such as excessive bronchial secretion or a partially closed glottis in the act of coughing, lifting, straining, or on playing wind instruments. Sir William Jenner was of the opinion that the less protected and less supported vesicles yield, dilate, rupture, and coalesce more or less in this manner, according to the amount of inflation, bronchial obstruction, and expulsive effort.

3. *Nutritive Change Theory.*—Cohnheim and others thought that there is probably a congenital pulmonary weakness, in consequence of a defective development of the yellow elastic tissue in the lungs, favoring dilatation and distention on extra exertion. Villemin is of the opinion that there is a hypertrophic development of the vesicular septa causing increased capacity. Auld inclines to the opinion that emphysema is due to a trophic neurosis of the terminal endings of the nerves in the lungs causing relaxation of the air vesicles. Brown-Séquard looked upon the disease as a nervous disturbance of the pneumogastric filaments. Walshe is of the opinion that the change in the air vesicles may be due to a primary nutritive change in the walls of the cells or in consequence of inactivity of the neighboring parts of the lung. Niemeyer was strongly of the opinion that the direction of the expiratory force is an etiological factor in the production of emphysema. For instance, in coughing or straining, the chest is fixed and the diaphragm is forcibly contracted, pushing the air in the lungs obliquely upwards, hence the upper parts and unprotected edges are the first to suffer.

In *acute general emphysema* the most prominent etiological factor is probably an extensive bronchitis, which causes more or less obstruction to the expulsion of air in the bronchi and bronchioles. This in turn produces an overdistention of the air vesicles which (if the cause be not removed) dilate or rupture. If this inspiratory expansion or obstructive expulsion continues, a general fibrosis with dilated, expanded, or ruptured air vesicles results and general vesicular emphysema is established.

Chronic hypertrophous emphysema most frequently results from chronic bronchial catarrh and asthma; the constant forcible expulsive efforts in coughing, with the partial plugging of the bronchi and the efforts to throw off the secretion, produce general vesicular enlargement throughout the lung with a slow interstitial hyperplasia. The vesicles lose their elasticity; the chest enlarges and rises to make room for the overdistended lungs, and a substantive emphysema is established.

Localized emphysema, both *acute* and *chronic*, is probably produced by expulsive efforts in obstructed parts of the lung. In the act of coughing, lifting, straining, etc., the glottis is closed and the expiratory pressure being greatest in the unobstructed lobules (because so many are closed or in an atelectatic state), the alveoli yield, especially in the unprotected parts of the lung, and localized emphysema results.

Atrophous or *senile emphysema* occurs in degenerative systemic changes and in advanced life. The septa, for the want of proper nutrition, from atrophy, pressure or dilatation, waste and become obliterated and the vesicles coalesce. The lung degenerates and atrophies, becoming smaller and smaller; the chest walls sink, and the lung capacity is reduced.

Pathology.

The lungs in *acute general emphysema* are universally distended. When the thorax is opened they protrude beyond the limits of the chest; they do not collapse, and they retain the impression of the finger. The lung tissue appears pale and the capillary network stretched and enlarged. There is usually evidence of extensive bronchitis and obstruction of the bronchi from the accumulation of the secretions. The air vesicles are nearly all enlarged and distended, but seldom ruptured or coalescent as is seen in the chronic form of the disease.

The lungs, in *chronic hypertrophous emphysema*—the most usual form of the disease—are greatly distended, covering the precordial region and bulging out of the thorax when this cavity is opened. The apices, anterior borders, and lower margins, representing the

unsupported parts, are often very much distended, especially if the disease is of long duration. The lung is pale, bloodless, collapses imperfectly, and presents a dry parchment-like feeling instead of the moist crepitation of a healthy lung. The vesicles are enlarged, many of them to the size of a pea or a bean. Frequently many of the lobules coalesce, forming irregular spaces or bullæ the size of a walnut. Contiguous lobules are thrown together, their septa remaining as withered threads or they may be entirely obliterated. The tissues often present spots of pigment, some of which are derived from the hæmoglobin of obliterated capillaries, and others, of a dark color with irregular angles, are due to the inhalation of carbon in the smoky atmosphere of large cities.

The vesicular septa are obliterated in consequence of the pressure of air in the vesicles producing distention with thinning of the walls, which eventually atrophy from a want of proper blood supply.

Structural alteration and fibrous formation are superinduced by the irritation of the air pressure, causing congestion in enlarged, toughened fibrous vesicles (Jenner). These sooner or later undergo fatty degeneration and absorption with obliteration of the septa (Rainey). Primary malnutrition of the pulmonary tissue leads to vesicular degeneration in chronic emphysema (Waters). The elastic tissues and capillaries degenerate, become absorbed, and disappear.

The earliest anatomical change consists in enlargement and division of the nuclei in the walls of the air cells, in consequence of the stimulus of stretching. The cell division and proliferation go on to granular disintegration and absorption. The cell granulation progresses from the endothelial lining of the air cells in the acini to the connective-tissue cells beyond, until the vesicle is perforated with holes and the lobules coalesce, the capillaries and elastic fibres being the last to disappear. In long-standing cases interstitial fibroid changes occur about the lobule, especially in emphysema which does not depend upon bronchitis; even the nerve filaments have been seen to undergo this interstitial change which is followed by granular degeneration (Auld). On microscopic examination the walls of the acini are found to be exceedingly thin, the septa are broken down, and have disappeared entirely or left mere projections into the alveolar area. Threads of fibrous tissue may be seen stretching across the air spaces, or a pigmented streak will indicate the remains of an obliterated capillary. In other parts of the lung the air spaces may be thickened from hyperplasia.

Lobular or *local* emphysema has a morbid anatomy similar to the other form, depending upon the length of time it has existed. The apices and margins of the lung are usually the seats of the disease.

In *senile* or *atrophous* emphysema the lungs are much diminished in size. The pulmonary tissues are pale, but pigmented, dry, and inelastic. The air vesicles are enlarged and the septa, fibrous, and fibro-elastic tissues are atrophied.

Besides the pathology of the air cells themselves, emphysematous lungs frequently present many other morbid appearances, such as extensive bronchitis, bronchiectasis, pleuritic adhesions, atelectasis, fibroid phthisis, and tuberculosis. The heart is usually displaced, the right ventricle lying more horizontally on the diaphragm with the apex to the left and downwards. From the obstruction to the pulmonary circulation there is always more or less hypertrophy and dilatation of the right cavities of the heart with a tendency to incompetency of the tricuspid valves. As before remarked, in many cases of emphysema there is a fibrosis of the lungs and other organs which has much to do with the seriousness of the prognosis. The incompetent tricuspid leads to chronic passive congestion in the stomach, liver, kidneys, and gastro-intestinal tracts, producing nutmeg liver, albuminuria, œdema, anasarca, and the long train of symptoms which lead to granular and fatty degeneration of the viscera.

Essential Morbid Anatomy of Emphysema.—

1. The lung contracts very little if any when the chest is opened; frequently it expands beyond the cavity; 2. The air spaces are much enlarged; the apex and margins frequently present large bullæ; 3. The elasticity of the lung, which assists in expiration, is much reduced or absent; pitting remains on pressure; 4. The vascular area available for aërating the blood is greatly diminished; 5. The lung is usually much enlarged in size but lighter in weight than normal; either bronchitis, bronchiectasis, pleural adhesions, or pulmonary fibrosis is usually present; 6. The pulmonary and other arteries often show signs of atheromatous degeneration; 7. The right heart is usually hypertrophied and dilated and there is tricuspid insufficiency; 8. Chronic interstitial nephritis and nutmeg liver are frequently present; 9. The costal cartilages are often calcified; 10. Œdema or general anasarca is a not infrequent complication.

Symptoms.

The symptoms of emphysema depend largely upon the complications present in each individual case. Dyspnœa is usually one of the most constant and distressing symptoms; it is generally increased after a full meal or on slight exertion, such as going upstairs or climbing a hill. As the disease progresses, expiratory dypsnœa becomes more and more pronounced. A slight cold or bronchitis is

almost certain to make it worse and frequently sets up spasmodic asthma with all the distressing symptoms of inspiratory and expiratory dyspnœa.

Bronchitis is frequently present with cough, and there is then considerable expectoration; but there may be no secretion when the cough is dry. There is not much actual pain beyond a dragging and uncomfortable feeling experienced below the sternum toward the ensiform cartilage. Another concomitant symptom is the interference with the proper aëration of the blood. As a result of the changes in the air cells, many of the pulmonary capillaries become obliterated, and the right side of the heart is in consequence affected, becoming, as the case progresses, hypertrophied and dilated with more or less tricuspid regurgitation; general venous congestion ensues, leading to dyspepsia, malassimilation of food, passive hyperæmia of the liver and kidneys, albuminuria, dropsy, and serious organic degeneration.

Physical Signs.

Inspection.—In most cases of emphysema we can detect the disease with considerable certainty at the first glance. The patients are usually quite well nourished, and in the early stage of the disease they appear quite plump or even somewhat bloated. The neck is usually short and compressed, the sterno-cleido-mastoid muscles acting as auxiliaries to inspiration and standing out prominently. The inspiratory contraction of the scaleni may be frequently seen or felt. The inspiratory movements are almost always accelerated, inspiration is short and labored, the thorax is raised high and is rigid, and expiration is visibly prolonged. In the usual form, chronic hypertrophous emphysema, the thorax assumes symmetrical bilateral enlargement, the "large-lung" or "barrel-shaped" chest. The ribs become more horizontal, the intercostal spaces are wider at the top and narrower at the bottom. The costal cartilages are quite rigid. The thorax becomes almost circular in form, increasing its antero-posterior diameter, and on inspiration and expiration all the ribs seem to move as one solid piece. The sternum is often abnormally prominent. There is bulging of the infraclavicular and mammary regions. The shoulders are elevated and brought forward. The breathing is labored and all the ordinary and auxiliary muscles of respiration are called into play. On exertion dyspnœa becomes pronounced. The veins in the neck are often distended and pulsating, and the lips are blue, giving evidence of obstructed venous circulation. The apex impulse is often absent and an epigastric cardiac impulse is commonly observed. A line of distended veins may sometimes be seen

surrounding the body of the lower margin of the ribs, constituting the so-called emphysematous girdle.

Palpation.—The whole chest moves *en bloc.* The vocal fremitus varies according to the absence or presence of bronchitis, bronchiectasis, etc. The apex beat of the heart is often not perceptible in the precordial space or it may be felt to the left and downwards.

Mensuration.—The tape measure shows a greater circumference of the chest due to an increase in its antero-posterior diameter.

Percussion reveals an increased area of the pulmonary sound and decreased area of the præcordium. The note, especially over the apices, is hyper-resonant, the percussion sound being at times quite tympanitic. The pitch is lowered and the pulmonary quality is greatly diminished. The percussion note varies but slightly between inspiration and expiration. The liver is pushed downwards and can be felt below the ribs on the right side. The inferior border of the lungs is often one or two inches below the normal position of inspiratory inflation. The pulmonary resonance extends on both sides in the back to the level of the first or second lumbar vertebra. Dilatation and hypertrophy of the right ventricle of the heart may also be detected by percussion, although the lungs generally cover the precordial region. The frequent epigastric pulsations and undulating pulsating movements in the jugular veins may be regarded as certain signs of dilatation of the right side of the heart.

Auscultation.—The inspiratory murmur is short, feeble or scarcely audible, while the expiratory sound is greatly prolonged and harsh. If much bronchial disease is present there will be sonorous or sibilant râles according to the amount of secretion present, with exaggerated respiratory sounds of harsh quality. Vocal resonance may be diminished or exaggerated. The heart sounds are feeble and there is apex displacement. In long-standing cases there may be roughness or incompetence of the tricuspid valve. The vesicular murmur itself often undergoes modification in pulmonary emphysema; the sounds are generally exaggerated and rougher than normal, and they are frequently so modified by bronchitis, bronchiectasis, collapse, broncho-pneumonia, and cirrhotic tissues in the lung as wholly to conceal the emphysematous condition.

The diminution of expiratory pressure in emphysema may be measured with the manometer; the normal expiratory pressure of 110 to 130 mm. sinking in emphysema to 100 or even 80 mm. The spirometer shows a corresponding diminution of the vital capacity of the lungs; the normal lung capacity of about 3,500 c.c. falling to 2,000 or even to 1,000 c.c.

The last stage of emphysema is usually characterized by the ap-

pearance of serious disturbance of compensation of the heart. The closure of numerous pulmonary capillaries results in hypertrophy of the right ventricle and impairment of the pulmonary circulation. The cyanosis which exists in most patients is due to incomplete oxygenation and to the blood stasis which extends backward from the right side of the heart into the veins of the body. As the disease progresses, however, the right ventricle becomes more and more feeble and stasis of the veins increases. There is serous transudation in the extremities and cavities of the body, and after long suffering the patient usually succumbs to anasarca with heart failure.

Diagnosis.

General chronic emphysema may occur at any time after adolescence. From the history of attacks of bronchitis, dyspnœa, and the physical signs of symmetrical enlargement of the chest, prolonged expiration, etc., the diagnosis of emphysema is comparatively easy, especially in the last stages of the affection when there are distinct signs of heart disease, although there may be danger of error when the prominent symptoms point to the heart or kidneys.

Differential Diagnosis.—Pneumothorax, hypertrophy of one lung, pleuritic effusion, chronic pneumonia, and excessive development of the lungs in athletes might be mistaken for emphysema at first glance; but when it is remembered that pneumothorax comes on more or less suddenly, affects only one side which is immovable and bulging, and is marked with tympanitic resonance and absence of respiratory sounds, vocal resonance, and vocal fremitus, while emphysema is bilateral and presents different physical signs, a mistake is not likely to occur.

Hypertrophy of one lung is rare; there are here normal vocal fremitus, resonance, and vesicular breathing; the relation of inspiration to expiration, as in health, is one to four, whereas in emphysema it is four to one.

In pleuritic effusion we have the disease on one side or at the base on both sides. There is diminution or absence of respiratory murmur, of vocal resonance, and of vocal fremitus. There is the history of an acute febrile disease with pain, elevated temperature, bulging of the affected side, etc.

Chronic fibroid pneumonia is often more or less associated with emphysema; the physical signs vary according to which disease is more pronounced.

Excessive development of an athlete's lungs can easily be distinguished from emphysema by the normal respiratory sounds, expansion, etc., and the physical condition of the patient. In localized

emphysema more care and attention to the physical signs are required. It must be distinguished from phthisical cavities and bronchiectasis. Atrophous emphysema is usually found in old people and is associated with a small chest and obvious senile decay.

Prognosis.

Emphysema in the acute form, like that resulting from whooping-cough, acute capillary bronchitis, and analogous diseases, may be cured if not of too long standing. Chronic emphysema must always be looked upon as a serious disease. Patients suffering with it may, with care, live many years, but any intercurrent disease such as bronchitis, pneumonia, pleurisy with effusion, phthisis, endocarditis, or pericarditis may produce fatal results at any time. Emphysema by itself seldom proves fatal. There are, however, a few cases in which there are progressive emaciation, anæmia, headaches, sleeplessness, and finally delirium, dyspnœa, and death without any apparent pulmonary complication. In these cases death is probably due to general arteriosclerosis and chronic Bright's disease. The duration of the disease depends entirely upon the intensity of the symptoms, the complications, the resistance of the patient, and upon what can be done in the way of prophylaxis. With care patients suffering with emphysema may be made comfortable for years, but with the first symptoms of respiratory and cardiac insufficiency the condition may become extremely dangerous.

Treatment.

Prophylaxis.—The treatment of emphysema must be largely one of preventing the occurrence of any pulmonary or bronchial disease. One attack of bronchitis renders the patient more susceptible to subsequent attacks, each one of which produces more and more emphysema. Pneumonia in an emphysematous lung often proves fatal, so may general bronchitis, overwork, straining, and lifting. The patient should move to a warm, moderately dry, equable climate, free from dust and winds, near the seashore. Many parts of California from Lake County to San Diego can be particularly recommended. Woollen undergarments should be worn and exercise should be taken with moderation. All causes known to aggravate the disease must be studiously avoided. The food should be well prepared and thoroughly digested. It is of prime importance to keep the alimentary canal in order and to supply more fuel than there is waste. Saccharine and amylaceous foods should be avoided, and alcohol and tobacco should be used sparingly if at all, and all excesses should be shunned.

Curative Treatment. The medicinal treatment of emphysema depends entirely upon the condition of the patient. There is no specific remedy for the disease. Of all remedies in ordinary cases, strychnine, gr. $\frac{1}{20}$ to $\frac{1}{10}$ in twenty-four hours in divided doses, is, perhaps, the most beneficial. If anæmia and loss of tonicity are present, iron and phosphorus may also be used. Excellent results have been obtained by the use of the pneumatic cabinet. The patient exhales into a rarefied chamber and thus empties the lungs of more complemental air than is expired under ordinary circumstances. I have found that the inhalation of compressed air and oxygen is likewise beneficial. Mechanical assistance has been recommended by Gerhardt. An assistant compresses the thorax systematically with his hands, twenty times per minute, for ten minutes, two to four times daily. This assists the expiratory movement and empties the lungs more completely than ordinary exhalation, and may assist in overcoming the relaxed condition of the air cells.

Any intercurrent disease, such as bronchitis, asthma, pneumonia, cardiac disease, venous congestion, hepatic and renal complications, must be met energetically by appropriate remedies. The usual chronic bronchitis is exceedingly troublesome and requires special attention to keep the patient comfortable. For this purpose I use with considerable success steam atomization of creosote, oil of cinnamon, and oil of eucalyptus, as follows:

℞ Creosote,
Oil of cinnamon,
Oil of eucalyptus, ... āā ʒ ij.
Glycerin, ... ʒ i.
Cologne water, ... q. s. ad ʒ xij.
Mix and employ in steam inhalation three times daily.

The patient is to live in a temperature which is never below 60° F. At night he should have the air in his sleeping apartment saturated with this inhalant.

Internally, the balsams of tolu, cubebs, or copaiba, terebene, or terpin hydrate with grindelia, lobelia, and syrup of codeine will be found useful.

In the chronic bronchitis of emphysema the following has proven beneficial in my hands:

℞ Oil of eucalyptus,
Extract of pinus canadensis, ... āā ʒ i.
Syrup of poppy,
Syrup of tolu, ... āā ʒ i.
White vaseline, ... q. s. ad ʒ vi.
Mix and make an emulsion. One tablespoonful after meals and at bedtime.

Attacks of dyspnœa and asthma due to spasmodic contraction of the bronchi are relieved by remedies which will relax the spasm and produce secretion from the tubes, such as nitroglycerin, stramonium, lobelia, iodide of potassium, and syrup of poppy. Constriction of the arteries requires nitrite of amyl, nitroglycerin, or other vaso-dilators.

It is important to keep the heart and blood in as good condition as possible. For this purpose strychnine, ferruginous tonics, convallaria, strophanthus, digitalis, and the phosphates are indicated.

Any disease of the antrum or nasal hyperæmia, catarrh, polypi, deviation of the septum, etc., had better be remedied, as attacks of dyspnœa and asthma may be produced by many of these conditions. Complications on the side of the alimentary canal, liver, kidneys, or other organs should be attended to on general principles.

Interlobular or Interstitial Emphysema.

Etiology.—The infiltration and accumulation of air in the interstitial or connective tissue of the lungs is most frequently seen in young children during an attack of whooping-cough, capillary bronchitis, broncho-pneumonia, croup, or convulsions, or after traumatism, such as tracheotomy.

Pathology.—Interlobular emphysema takes place only in young children because as the lung develops the intervals of connective tissue between the lobules disappear. Hence adults do not have interstitial pulmonary emphysema. During a violent expiratory paroxysm some of the delicate air cells rupture and air escapes into the connective tissue surrounding the lobules or under the visceral pleura, where it may occasionally be seen post mortem as strings or beads of air bubbles. These may be displaced by pressure. The amount of emphysema present varies. At times only a few lobules are surrounded by air beads, and again the air may extend along the connective tissue to the roots of the lungs and diffuse itself into the mediastina, neck, face, and general cutaneous surface. When interlobular emphysema occurs in the adult it is usually the result of an injury. Occasionally the pleura may rupture and cause pneumothorax.

Symptoms.—Sudden and severe dyspnœa after a paroxysm of coughing in a child is strongly symptomatic of interlobular emphysema. Occasionally faint friction sounds may be heard. On rare occasions pneumothorax and general cutaneous emphysema may occur with the usual symptoms and signs.

Treatment.—The spreading of the emphysema should be checked by keeping the patient perfectly quiet and by using precautionary

measures in preventing paroxysmal cough or other cause of the disease. The cough may be loosened by expectorants, relaxants, or the inhalation of suitable remedies in steam. In other words, the cause should be treated and the patient's strength supported. Carbonate of ammonium, Hoffman's anodyne, whiskey, digitalis, or strychnine may be indicated.

ATELECTASIS.

Definition.—Under the head of atelectasis it is well to clearly understand that in *atelectasis proper* the lung has never been inflated, hence it is a *fœtal* or *congenital condition.*

In *apneumatosis*, on the other hand, the air cells have collapsed from some obstruction or compression of the bronchi or lung tissue, and the collapse is an *acquired* condition.

Etiology.

Atelectasis proper, or congenital collapse of the lung, is caused by the inability of a child when born to expand the air cells. This condition is seen in very weakly children—strumous, rickety, or syphilitic—whose respiratory movements are insufficient to draw in the necessary amount of air to dilate all the air cells in the lungs. Apneumatosis or acquired collapse is caused by some *obstruction* such as chronic enlargement of the tonsils, adenoid vegetations in the naso-pharynx, tumors, growths, cancer, aneurysm, etc., within or without the naso-pharynx, larynx, and bronchi. Viscid mucus and purulent secretion, as in diffuse capillary bronchitis, etc., may also cause collapse of portions of the lung. Collapse of the lungs may also be due to compression as in pleural effusion, hypertrophy of the heart, pericardial effusion, aneurysms, mediastinal tumors and growths, angular curvature of the spine (kypho-scoliosis), goitre and growths in the neck, abscesses of the liver, diaphragmatic growths, abdominal tumors, ascites, in short, anything which operates as a mechanical obstruction to the entrance of air into the cells or anything which compresses the lungs or portions of them to such an extent as to preclude the possibility of lung expansion. Unless the atmosphere has free access to the air cells, imprisoned air in the lungs soon becomes absorbed and the air cells collapse. It is important to remember that patients long ill, as in typhoid fever for instance, who lie in the dorsal position for weeks at a time, may be found to have collapse of the lower lobes posteriorly when they attempt to sit up. Plugging of the smaller bronchioles in capillary bronchitis in chil-

dren and old people must not be overlooked as an etiological factor of pulmonary collapse.

The simplest form of acquired collapse of the lungs is observed in cases in which the lung is compressed by an accumulation of fluid within the pleural cavity. The lung is much reduced in size, its pleural surface is livid, and its tissue is uniformly smooth, dark, and flesh-like on section—all the structures, air vesicles, bronchi, and blood-vessels being alike compressed; the tissue is heavier than water, because all the air has been squeezed out of it, and yet it is capable of being reinflated. A second variety of atelectasis is frequently observed in patients suffering with bronchitis, fevers, and other affections in which respiration is incompletely performed; here the collapse occurs in scattered groups of lobules which are most abundant near the lower portions of the lungs. These lungs present depressed, livid-colored patches, alternating with elevated portions of comparatively normal lung tissue. On section the depressed areas are found to correspond to dark-colored, airless, tough, smooth, flesh-like patches which extend variously in depth into the substance of the lung. The immediate cause of the production of acquired collapse of the lungs seems to be, as generally accepted, the plugging more or less completely with mucus of the smaller air passages in the lungs; little plugs of mucus acting like a valve will permit of complete expiration, but may prevent the entrance of air into the vesicles.

Pathology.

The appearance of the lungs in collapse or atelectasis is very characteristic. The lung tissue has a violet or purplish-gray color. It is, of course, airless and non-crepitant. It is *dry* on section and more or less tough or fibrous to the touch, having undergone the so-called splenification or carnification. Such lung tissue sinks in water. Frequently an apneumatic lung will present dark-grayish, depressed patches on its surface, varying in size from that of a pea to that of a walnut, marking the site of the collapsed air cells. At first the collapsed lobule is congested; but owing to pressure, the blood soon coagulates in the vessels and then becomes decolorized and absorbed, leaving the vessels completely obliterated. The walls of the collapsed alveoli after a time become agglutinated and not infrequently a bronchopneumonia is set up. The base of the left upper lobe and the middle of the right lung are the most frequent seats of atelectasis; next in order come the posterior parts of the upper and lower lobes on both sides. The superficial lobules are much more frequently affected than the parts which lie deeper in the lung. Collapsed lobules can

usually be blown up post mortem by means of a blowpipe introduced into the communicating bronchus. The lung tissue, however, soon subsides unless the bronchus is tied. The tubes leading to the collapsed lobules are frequently found to contain viscid or purulent secretion, blocking the free access of air. Other portions of the lung are often found to be emphysematous on account of the obstruction to respiration and the diminished air capacity of the lungs.

The precise pathological characteristics of an atelectatic lung depend somewhat on the duration of the collapse, the amount and condition of the blood in the affected lobule, and the changes which have taken place in the surrounding tissues. The collapsed lobules on section are quite smooth, more or less pyramidal in shape, with the base extending outwards. The size of an atelectatic spot depends generally upon the size of the bronchus obstructed; the color will also depend measurably upon the length of time it has been collapsed. The degree of congestion, the bulk, the firmness, and the difficulty of reinflation all depend largely upon the amount of contraction which has taken place in and about the lobules. The tubes leading to the collapsed lobules are generally found to contain a viscid or semi-solid secretion obstructing the air passage and in the majority of cases probably causing the collapse.

Symptoms.

In congenital atelectasis the infant is weakly and puny, presenting a more or less livid color, having shallow breathing and a feeble cry. With each inspiration the lower part of the chest walls is drawn in and the intercostal spaces are usually much depressed. The breath sounds are feeble or absent in the parts affected and death is a frequent termination. In acquired collapse or apneumatosis the condition is usually lobular in extent, presenting absence or diminution of vocal fremitus, and vesicular murmur, dulness on percussion, and localized bronchial breathing. There may also be present moist or dry subcrepitation from the adhesion of the air cells and large moist râles from the bronchitis. There are usually present dyspnœa, quick shallow breathing, feeble and ineffectual coughing, with signs of deficient aëration of the blood and considerable wasting and exhaustion.

Owing to the fact that atelectasis of the lungs frequently is associated with emphysema, bronchitis, and other morbid processes, the physical signs are not always diagnostic. The inspiratory dyspnœa, in which the lower part of the chest wall retracts during inspiration, is one of the typical signs of atelectasis. The cyanosis from

deficient blood aëration and the marked wasting and exhaustion are important points to consider besides the impaired resonance and weak breath sounds. In atelectasis from compression, as in pleurisy with effusion, etc., the symptoms would be those of compression and consequent dyspnœa rather than those referring to any special disease to the lungs themselves.

Diagnosis.

Congenital atelectasis is diagnosed by the weakly condition of the child, dyspnœa, feeble cry, retraction of the lower part of the chest walls, and absence of vesicular murmur. The conditions of the child are those of improper aëration of the blood and deficient lung expansion. Such children are apt to be rickety, scrofulous, syphilitic, or born of intemperate and sickly parents. In acquired collapse the diagnosis depends on the etiological factor of obstruction or compression. When pleuritic effusion or enlargement of the heart or of some of the abdominal organs causes the compression, the diagnosis is easy; but when alveolar collapse occurs from bronchitis or mediastinal growths the diagnosis is not so apparent. By careful auscultation it is possible, however, to determine (as a rule) the extent of the disease. It is important in this condition particularly (as in all diseases generally) to discover the cause operative in the production of the affection.

Prognosis and Treatment.

In the congenital form of the disease the prognosis is unfavorable, unless the child, by supporting, stimulating, and specific treatment, can be made strong enough to expand its lungs. The struma, rickets, or syphilis should be treated on general principles. In the acquired form of collapse the primary cause must be discovered and removed if possible, and deep inspirations in rarefied air or in air chambers charged with oxygen should be practised. In long-continued diseases the patient should be turned frequently from side to side. Deep inspiratory efforts should be encouraged. Further treatment must be supportive and symptomatic. Stimulating expectorants will assist in clearing the bronchial tubes and forced inspiration will dilate the air vesicles. Friction with some stimulating liniment and poultices, sinapisms, or cupping may assist toward the establishment of the circulation in the collapsed lobules. Tonics of iron and strychnine with the best of food and fresh air will do much to reinflate the collapsed lobules, and the removal of the causes of compression such as pleuritic effusions, abdominal fluids, etc., will

assist in establishing normal respiration in the atelectatic lung. When pulmonary collapse is suspected during the course of an attack of bronchitis in children, one of the main objects in treatment should be to promote the liquefaction of the secretion, and to assist the expulsive efforts to rid the tubes of the sputum. For this purpose ammonium chloride, serpentaria, sanguinaria, and eucalyptus have proven very efficacious. When the first-named agent does not liquefy the secretion rapidly enough, hot applications to the chest, hot demulcent drinks, the inhalation of steam, and the administration of a little iodide of potassium often succeed admirably. When the sputum is difficult of expectoration, caution should be exercised in employing the ordinary cough remedies containing opium or any of its derivatives, as the secretion will become more solid instead of more liquid. In chronic cases the balsam of cubebs or of copaiba, terebene with cod-liver oil, or petroleum emulsion have been found serviceable.

LOBULAR PNEUMONIA.

Definition.—This disease, called also broncho-pneumonia, disseminated pneumonia, or catarrhal pneumonia, is now regarded by most writers as an infectious inflammation, the pathogenic micro-organism of which is supposed to be either the bacillus of lobar pneumonia or the streptococcus of suppuration. Lobular pneumonia is that form of inflammation of the parenchyma and air vesicles which occurs in isolated patches comprising one lobule or a number of adjacent lobules in different parts of the lungs. The disease in the vast majority of cases begins in inflammation of the bronchial mucous membrane, which finally involves the air cells and parenchyma of the lungs.

In all forms of pneumonia more or less bronchitis is necessarily associated with the disease. In broncho-pneumonia there is always some capillary bronchitis present. In lobar pneumonia the etiological factor, probably the pneumococcus, seems to enter the air cells themselves, setting up at first hyperæmia, then inflammation with exudation. In lobular pneumonia it has always been held that the inflammation at first is in the bronchial mucous membrane and afterwards extends into the air cells and perilobular tissues. Recent writers, however, declare that the inflammation is caused by a micro-organism, and that it does not involve the mucous membrane of the bronchial tubes, but originally affects the air cells themselves. The writer is in doubt as to the correctness of this view, because it is of daily occurrence that a bronchitis, pure and simple, extends into a capillary bronchitis and that a capillary bronchitis extends into the

air cells, producing broncho-pneumonia. If catarrhal pneumonia depends upon the presence of the pneumococcus, we should be able to show that capillary bronchitis and many cases of ordinary bronchitis must also depend upon a microbe; in the mean while we rather incline to the view that lobular pneumonia is an extension of the inflammatory process present in the capillary mucous membrane which involves the air cells and tissues immediately surrounding them.

There are three varieties of lobular pneumonia:

1. Simple or acute lobular pneumonia, following a severe case of catarrhal bronchitis; 2. A secondary form which comes on after whooping-cough or some other febrile infective disease; 3. Embolic or suppurating infective disseminated pneumonia, which occurs at times in the course of infective endocarditis, pyæmia, and malignant fevers.

Etiology.

Lobular pneumonia is probably caused by a direct extension of the inflammatory process along the bronchioles to the air cells, setting up pneumonia of individual lobules or sets of air vesicles. It may occur as a primary inflammation, but is most frequently met with as a secondary affection in connection with whooping-cough, measles, scarlet fever, diphtheria, influenza, and simple and capillary bronchitis in children. It is also seen in grown people after exposure to cold, and in cases of influenza, typhoid fever, small-pox, erysipelas, and emphysema. Broncho-pneumonia is also met with in connection with bronchiectasis, chronic bronchitis, asthma, cirrhosis of the lungs, and phthisis. People long confined in bed from some injury, children of a weakly or strumous diathesis, and those poorly nourished are most liable to contract the disease. Rickets and chronic diarrhœa predispose to this form of pneumonia, as does any debilitating cause existing either in children or in adults. The habitual breathing of vitiated air, the long-continued dorsal position, senile debility, and chronic diseases often determine an attack of catarrhal pneumonia which may prove fatal. Many cases of pneumonia occurring in aged persons after exposure to cold or in the subjects of continued fevers, acute and chronic diseases generally, are undoubtedly of the broncho-pneumonic variety. These often terminate fatally. Malignant fevers, malignant endocarditis, and pyæmic processes generally are prone to set up embolic lobular pneumonias which frequently terminate in death.

Pathology.

The usual pathological conditions found in lobular pneumonia are catarrhal in nature. There is no fibrinous exudation. The alveoli are filled with epithelial elements, leucocytes, and mucoid secretion from the bronchial tubes. The mucous membrane of the bronchioles leading to the invaded air cells is thickened, softened, and swollen, of a dark red-color, and covered with secretion. The small tubes and alveoli are often so entirely choked with inflammatory products and epithelial débris as to prevent the entrance of any air whatever. The peribronchial and perivesicular tissues present interstitial inflammation with numerous leucocytes and enlarged capillaries. The bronchial glands are often enlarged and softened. The inflamed lobules are irregularly disseminated throughout both lungs, especially toward the bases and along the lower and free borders at or near the pleural surface. The inflamed areas vary considerably in size, from that of a pea to that of a hazelnut. When superficial they are of pyramidal or wedge-like form with their bases toward the pleural surface. These superficially inflamed lobules project somewhat beyond the pleural surface and feel firm and hard to the touch. In reality they consist of the pleural membrane and interstitial tissues of the alveoli which are distended with the catarrhal débris and muco-purulent deposit. Slight pressure suffices to break them down, whereupon a whitish, opaque, non-frothy, muco-purulent fluid exudes. The bronchioles leading into the inflamed lobules are frequently dilated and contain purulent fluid. The tissues of the inflamed pneumonic spots sink in water. In favorable cases the cells, débris, and fluid undergo fatty changes which enable them to be absorbed or liquefied and expectorated. When catarrhal or lobular pneumonia follows lobular collapse the inflammatory process and morbid appearances are generally confined to isolated lobules in different parts of the lung; but when the inflammation progresses and the adjacent lobules are involved and coalesce, larger areas of pneumonic spots are formed. If the inflammation is severe, septic, or pyæmic in character, abscesses form or caseous degeneration ensues, which frequently leads to extensive destruction of lung tissue and the formation of cavities, gangrene, or chronic interstitial pneumonia. Broncho-pneumonia, which is independent of lobular collapse, often presents numerous small ill-defined whitish-yellow spots scattered throughout the lung, yielding an opaque milky fluid on pressure.

Lobular pneumonia is generally associated with acute bronchitis, or secondary bronchitis from whooping-cough, scarlet fever, measles,

or some chronic disease or general senile debility, and there is usually present more or less pulmonary hyperæmia, œdema, and collapse of the lobules.

Lobular pneumonia is mostly confined to young children and old people. In a typical example the lung is studded with pneumonic patches varying in size from that of a pea to that of a marble, which involve one or more pulmonary lobules. The lobules are separated from one another by a network of healthy and still crepitant lung tissue. True lobular pneumonia is nearly always secondary to the blocking up of the air passages and especially those of the capillary tubes. The capillary secretion may be sucked into the air cells themselves and thus set up the lobular pneumonia, or the air cells may become involved by direct extension from the mucous membrane of the bronchioles. Whatever view we take of broncho-pneumonia it is certain that we find in the inflamed parts not only overgrown and modified epithelial cells, but also, according to the various stages of the disease, much granular material and many embryonic cells. The dependence of lobular pneumonia upon some obstruction of the tubes is further shown by the fact that lobular collapse is often associated with it.

Disseminated pneumonia is closely related to lobular pneumonia, but the pathology is different; in disseminated pneumonia the obstruction is not in the capillary bronchial tubes, but in the capillary branches of the pulmonary artery, and consists of an embolus or a thrombus. In pneumonia supervening in the course of pyæmia the pulmonary capillaries are choked by septic emboli which set up localized congestion, inflammation, and suppuration. There are small patches scattered about the lungs, most marked toward the bases and toward the surface of the lungs, which may be in various stages of hyperæmia, simple engorgement, or red and gray hepatization. These pneumonic spots, especially in the embolic variety, are very apt to undergo decomposition, setting up abscesses or gangrenous areas in the lungs. In lobular pneumonia there is apt to be considerable pulmonary congestion and œdema surrounding each affected lobule, and there is considerable danger of the coalescence of half a dozen or dozen lobules in the immediate vicinity.

Catarrhal pneumonia not infrequently leads to extensive consolidation of the lung resembling that of croupous or lobar pneumonia, or it may become chronic and last for months, leaving dilated bronchi and thickened walls with peribronchial changes which progress toward interstitial fibrosis or cirrhosis of the lung.

The post-mortem appearance of broncho-pneumonic lungs presents considerable variation. In some cases the mucous membrane of the

trachea and bronchi may appear normal or it may be congested. The small bronchi, however, nearly always show signs of inflammation and thickening and frequently contain pus. Around the thickened bronchi are found zones of consolidated lung tissue varying in size from that of a single lobule to that of three or four. It will also be observed that there are scattered about throughout the lungs irregular patches of consolidated lobules irrespective of any bronchial involvement. In many cases the lobular consolidation extends toward the surface and involves the pleura, which is then generally coated with fibrin. In very young children the consolidated portions are frequently solid, due to collapse of the air vesicles forming areas of atelectasis. One of the essential lesions of broncho-pneumonia is inflammation of the walls of the bronchi as well as of the air cells and the parenchyma of the lungs immediately surrounding the inflamed lobules. Not only are the bronchial tubes thickened, inflamed, and infiltrated, but the walls of the air spaces are thickened. Their cavities are filled with connective tissue, detritus, and fibrin, pus, and epithelium. The inflammation seems to be at first not catarrhal or destructive but productive, that is to say, the cells granulate forming new ones, the whole tending to the formation of new tissue. The bronchial glands are usually much swollen and inflamed; many of the smaller bronchi may be dilated. There is always considerable œdema about the consolidated spots, frequently atelectasis and occasionally emphysema. There is always present considerable catarrhal inflammation of the bronchi. The lobules tend to fill up with inflammatory new product. The same productive inflammation extends to the pleura. When involved there is frequently dilatation and obstruction of the bronchioles.

The products of inflammation are not similar to those found in lobar pneumonia; they seem to be more fibrinous, having, as just remarked, a greater tendency to the formation of new tissue. A broncho-pneumonia, therefore, may continue for several weeks and be followed by cirrhosis of the lungs because the inflammatory material seems to have a tendency to organize new fibrous tissue, especially that which takes place outside of the bronchial tubes. The bronchial tubes themselves, the peribronchial tissues, and walls of the air cells are thickened, and this new formation may extend to the pleura so that it can readily be seen how an interstitial fibrosis of the lungs may result from a broncho-pneumonia.

Symptoms and Physical Signs.

Broncho-pneumonia is usually secondary to acute general bronchitis extending by continuity into the air cells, or it follows measles,

scarlet ever, diphtheria, influenza, whooping-cough, etc. The fever which often rises to 104° and 105° F., is usually preceded by a chill of short duration or a succession of chills as different lobules or parts of the lungs are invaded. This pyrexia is one of the most important signs of the disease. These peculiar chills and elevation of temperature with frequent remissions and exacerbations are indicative of the extension of the disease. The skin is moist and perspiring. The pulse increases in frequency with each exacerbation and sometimes becomes feeble and irregular. There are rapid breathing, cough, dyspnœa, restlessness, and frequently cerebral symptoms. There is often considerable pain in various portions of the chest corresponding to the lobules involved. The expectoration is simply catarrhal and not bloody. The disease may last two or three weeks and ends by lysis, never by crisis. On examination, the consolidated portions of the lung will give increased vocal fremitus, deficient resonance, bronchial breathing with fine scattered crepitation, or crackling râles, bronchophony, and, when extensive bronchitis exists, the usual large moist râles of bronchial cough. One can frequently follow bronchial breathing with a stethoscope into the air cells and there find subcrepitant râles or a consolidated lobule. The surrounding lung tissue appears perfectly healthy. This is characteristic of broncho-pneumonia. Several of these spots from the size of a ten-cent piece to that of a silver dollar can be discovered by careful auscultation. At times several of these broncho-pneumonic spots may coalesce and then a lobar pneumonia is set up with consolidation, rusty sputa, etc.

In very young children the only observable symptoms are convulsions instead of chills, fever, great prostration, and rapid breathing. There is seldom much cough and the special physical signs are often difficult to determine. If the disease be at all extensive, the bronchioles are closed by the inflammatory product, a great number of lobules are involved, and death generally takes place in a few days. In older children when the disease is secondary to whooping-cough, measles, scarlet fever, pharyngitis, laryngitis, or bronchitis of the larger tubes, the prognosis is more favorable. There may be a chill or there may be a convulsion at the onset of the disease, after which the temperature rises rapidly. There may be vomiting and delirium with great oppression of breathing, great restlessness, and considerable cough. In ordinarily severe cases of broncho-pneumonia the child's face is flushed, the tongue is coated at first with a white, heavy fur which soon becomes dark and dry. There is often vomiting and diarrhœa, the temperature remains between 99° and 105° F., with frequent remissions and exacerbations. There is considerable pain

over the chest, together with constant cough in paroxysmal attacks, and great restlessness; the child tosses from one side of the bed to the other and with difficulty takes nourishment. The urine is scanty and high-colored, the skin, however, is usually moist and perspiring. It is well to remember that children up to the age of three or four years swallow the sputum instead of spitting it out. This may have something to do with the vomiting and diarrhœa. The duration of broncho-pneumonia in children varies considerably in different cases. In the fatal cases of very young children many die within two days, most within the first week. The great danger lies in the involvement of new lobules. The case may be going along very satisfactorily, the temperature may be 99° F. or normal, the pulse good, the child quiet and resting easily when within a few hours the temperature will be up to 105° F., with all the symptoms of pyrexia, restlessness, coughing, delirium, etc., so that in any given case it is impossible to prognosticate the condition of the child a day or a week hence. Many cases drag on with remissions and exacerbations for two, four, or even eight weeks and still make perfect recoveries. Resolution, as before mentioned, always takes place by lysis, and the period for complete restoration after the last chill or the last pyrexia is from one to two weeks. In most cases of broncho-pneumonia in children the temperature oscillates between 99° and 104° F., and the respiration varies from 40 to 80 per minute with a pulse of from 120 to 170 per minute; yet it is possible for a child with a temperature of 105° F., pulse 170, and respiration 80 to recover perfectly in fourteen days from the time of the last exacerbation. In a certain number of cases the cerebral symptoms are such as to lead one to suppose that the meninges of the brain are involved; there are convulsion after convulsion with intervening restlessness and delirium, moving of the head from side to side, oscillation of the eyes, high fever, and great prostration with loss of appetite, vomiting, and all the symptoms of meningitis. These cases are not so apt to recover unless they improve within a few days. Whenever there is any strumous diathesis present or a tendency to tuberculosis, the patient does not progress so favorably; tubercular meningitis is apt to supervene, the lungs sometimes become more congested, there is greater prostration, continuous high temperature, poor appetite, great loss of flesh, considerable cough, and every now and again the lymphatic glands will be observed to be enlarged, giving evidence of the strumous character of the disease and indicating that the bronchial and pulmonary glands are involved as well. In adults broncho-pneumonia presents several different symptoms. There is generally a distinct chill or a succession of chills with considerable headache, pains in the back and

chest, some vomiting followed by great prostration, high temperature, rapid pulse, and rapid respiration. The temperature will run up to 105° F. The patient will be exceedingly restless, complain of pain in the chest and severe dyspnœa. The cough is at first dry, but in a few hours the expectoration will become profusely mucous and it may be blood-stained. There is great restlessness, generally sleeplessness, and considerable delirium. The urine is scanty and apt to become albuminous. The skin is moist and there is free expectoration. There may be vomiting and diarrhœa at the onset, but usually constipation comes on in the course of a few days. The disease lasts from ten to twenty days. It is not so apt to prove fatal in adults as in young children and very old people. It is not infrequent in these cases of lobular pneumonia in adults to leave the patient comfortable during the morning hours with a temperature of about 99° or 100° F., breathing easily, appetite fair, and expressing himself as being comfortable, and to find, a few hours later, another chill with great elevation of temperature and all the symptoms of another attack coming on. This simply means the involvement of new areas or new lobules in the lung, and of course each exacerbation renders the prognosis more grave. In old people broncho-pneumonia is also exceedingly serious. In them it generally comes on with slight chilly sensations and some pyrexia, perhaps to 102° or 103° F. There is considerable headache, with backache and pains in the limbs. The pulse is accelerated but soon becomes feeble, there is loss of appetite, coating of the tongue, and, generally, constipation. The skin becomes moist and clammy, the breathing oppressed, and the cough very troublesome. In most of these cases of broncho-pneumonia in the aged which have come under the observation of the writer, there has been considerable bronchitis, emphysema, bronchial asthma, or cirrhosis of the lung present at the time of the attack. The catarrhal pneumonia seems to involve many points in the lungs. Crepitation is generally heard on the second or third day. The sputum in these cases is apt to be tinged with blood after the first three days, and the prostration is severe. These cases may do fairly well for five or six days or a week, when if other lobules are involved there are more chilly sensations, more febrile symptoms, and greater prostration. A fatal termination is almost inevitable. The physical signs in all these cases depend entirely upon the lung tissue involved and the stage of the involvement. There is always rapid and bronchial breathing, with the usual signs of bronchitis; there are also large and small mucous râles, lobular crepitation, and considerable cough, usually more or less paroxysmal in character. The sputum is generally mucoid but occasionally tinged with blood. When the lobular

nodules extend to the pleura in considerable numbers there is very apt to be set up a fibrinous or secondary sero-fibrinous or even a purulent pleurisy.

Diagnosis.

Lobular pneumonia is diagnosed by its mode of onset. It attacks those already suffering with extensive bronchitis, capillary bronchitis, whooping-cough, or adynamia from any cause. The disease is most frequent among children and old people. It begins by chilly sensations or a convulsion, during the progress of bronchitis or some of the acute infectious diseases. The temperature runs up rapidly to 103 105° F. There is then considerable paroxysmal coughing with mucous expectoration and great restlessness, the child tossing from one side of the bed to the other. In a day or two the patient may improve rapidly and the temperature may be down to normal, but in from six to twelve hours another exacerbation occurs with high temperature and great prostration. Several of these remissions and exacerbations are liable to recur at any time for a week or two and constitute one of the diagnostic points of the disease. In old people the disease runs a subacute course with exacerbations of fever, restlessness, anxiety, and prostration; loss of strength and wasting are prominent symptoms. In lobular pneumonia there are no rusty sputum, no severe and prolonged chill, no dry hot skin, and no extensive crepitation followed by consolidation as in the case of croupous pneumonia. There are not the great pain, pleuritic friction, and effusion observed in pleurisy. The exacerbations of fever, restlessness, and dyspnœa will indicate that it is not typhoid. The preceding bronchitis and obvious thoracic disease will indicate that the meninges of the brain are not the seat of the disorder. The sputum, regular elevation of temperature at night, apical infiltration, and the general condition of the patient will be sufficient data upon which to differentiate between bronchopneumonia and acute general tuberculosis. It is well to remember, however, that bronchopneumonia offers a good soil for the implantation of tubercle bacilli so that many cases sooner or later become consumptive. It must also be borne in mind that in measles, whooping-cough, scarlet fever, typhoid fever, malignant fevers, pyæmia, and many other acute and chronic diseases a bronchopneumonia may be set up.

Prognosis.

This depends largely upon the cause of the disease. In young children in whom there is extensive capillary bronchitis, and in old people lobular pneumonia is very prone to prove fatal. In ordinary sthenic cases following bronchitis the prognosis is good, and in many

cases that look almost hopeless recovery must not be despaired of. The possible association of catarrhal pneumonia with tuberculosis should not be lost sight of in giving a prognosis, and the liability to cirrhosis of the lungs is an important element as regards ultimate recovery.

Treatment.

The patient must be kept at rest in bed, in a *warm*, well-ventilated room. A large jacket poultice of flaxseed meal, applied hot every hour and covered with oil-silk, has proven of great benefit and comfort to the writer's patients. A stimulating liniment, such as one of equal parts of turpentine, camphorated oil, and ammonia liniment, applied after the first day or two is also of benefit. Ice is sometimes applied to the chest. The inhalation of vapor impregnated with eucalyptol or oil of cinnamon is desirable. The bowels should be freely opened with salines, but strong purgatives and all remedies that tend to lower the vitality of the patient should be avoided. Expectorants of ammonium carbonate, squill, senega, and tolu are to be given as required. Great caution is necessary in the administration of opium to young children. The heart must be constantly watched, as the disease at best is very depressing and weakening. Tonics such as grain doses of quinine, strychnine (gr. $\frac{1}{60}$), digitalis, and whiskey will frequently be called into requisition.

Several ounces of good liquid food such as broths, *home-made* beef tea, custard, rice, sago, tapioca, eggs, and milk should be given every three hours. Flaxseed tea (℥ ss. to Oi.) or gum arabic water (℥ i. to Oi.) flavored with a little fruit juice and a lemon will often prove refreshing and demulcent, assisting the expectoration. Whenever broncho-pneumonia comes on in the course of other diseases the original complaint must not be lost sight of, and yet it is likely that the chief attention must be paid to the lobular disease, as it is very fatal in exhausting, debilitated, and chronic affections.

Chronic Pneumonia.

Synonyms.—Interstitial Pneumonia, Cirrhosis of the Lungs, Induration of the Lung, Pulmonary Fibrosis, Fibroid Phthisis.

Definition.—By the expression chronic pneumonia, or cirrhosis of the lung, we mean an induration of the lung caused by development of nucleated fibroid tissue around the bronchial tubes, in the interlobular septa, or in the walls of the air cells. This overgrowth of connective tissue may occur at one or all of these situations and causes sooner or later the gradual effacement of the air cells.

Etiology.

The formation of an overgrowth of connective tissue in the lung is in many cases simply a local process, and it then appears to be compensatory. This is well shown in those cases where the new connective tissue tends to encapsulate and thus isolate morbid deposits and irritating substances from the rest of the lung, as occurs when tubercular deposits, hemorrhagic infarctions, abscesses, etc., are so surrounded. Any irritation sufficiently protracted will cause this change. It is, therefore, always present in, or supervenes upon, all forms of prolonged inflammation of the lung tissue; indeed, every case of pulmonary consumption, unless it runs an extremely rapid course, is accompanied by it. It is a constant attendant upon all kinds of morbid growth and the infective granulomata are its most potent etiological factors. In cases of acute pneumonia if resolution fails to take place or the affection assumes a more chronic form, the fibrous framework of the lungs will become involved, and proliferation of its elements will supervene. This is not an infrequent occurrence in the pneumonias of those children whose hereditary predisposition or whose poor nourishment and unhygienic surroundings render them susceptible to all kinds of retrograde processes. Lobar pneumonia is less frequently a cause than is bronchopneumonia. This is accounted for by the fact that the latter is more liable to be prolonged or to become chronic and that there is a frequent association of bronchiectases with it. These bronchiectases are also present in chronic bronchitis, especially when it is complicated by whooping-cough. In whatever way they originate they always favor the persistence of catarrhal and pneumonic processes, by rendering difficult the removal of the secretion which, therefore, accumulates and tends to keep up the irritation, both in the dilated bronchi and in the alveoli; and this persistence of the pulmonary irritation leads to induration of the bronchial and pulmonary wall. The new connective tissue in its turn tends to the formation of bronchiectases, and it is in this way that the process is gradually extended until in some cases large areas of the lung become involved. A chronic bronchitis may also cause connective-tissue development by exciting a chronic dry pleurisy with its concomitant adhesions and thickenings. Pleurisy with effusion, however, rarely causes fibrous growth; it is only in those cases which are very chronic and where the pressure on the lung has caused collapse that it is liable to occur, and even in these cases the process is only circumscribed. The irritation excited by the constant inhalation of certain kinds of dust such as occurs in miners, stone-cutters, potters, grinders, etc., always causes interstitial pneumonia. The continuous

irritation caused by the inhaled particles sets up a bronchial and alveolar inflammation which eventually involves the fibrous framework of the lungs. The embryonic tissue, no matter how it may originate, has a tendency to involve large areas and always eventually contracts, tending thus to obliterate the contained blood-vessels, bronchi, and alveoli.

By constriction or obliteration of the bronchial tubes, dilatations are nearly always formed; this is supposed to be due to various causes. It has been maintained that with the diminution in the size of the lung, there is compensatory retraction of the chest walls; but that this compensation, being inadequate to the loss in size, dilatation of the bronchia occurs to restore the equilibrium. In some cases it is perhaps the result of atmospheric pressure during inspiration; portions of the lungs being incapable of expansion, and other portions suffering compensatory dilatation. This is especially liable to occur during the violent inspiratory efforts that attend the fits of coughing which are so common in this disease.

Another important consideration in relation to the etiological factors of cirrhosis of the lung is that of general arteriosclerosis observed in chronic Bright's disease, in which a similar condition occurs in several organs at the same time, more especially the lungs, liver, and kidneys. The irritant condition of the blood in these cases seems to operate on the fibrous tissues in the blood-vessels themselves and through them on the fibrous tissues adjacent, setting up an irritation and a slow but continuous inflammation with the gradual formation of new fibrous tissue. When this formation of new fibrous tissue occurs around the bronchioles and air vesicles, the same law applies which induces all newly formed fibrous tissue to contract. The result is that the bronchioles are dilated, and the air vesicles are either dilated, contracted, or obliterated. The blood-vessels become obliterated and the parenchyma of the lungs becomes condensed. This etiological factor in the production of chronic pneumonia or fibroid phthisis seems to be generally overlooked, but I believe that it is nevertheless as potent a factor in the production of cirrhosis of the lungs as it is in the production of cirrhosis of the liver or cirrhosis of the kidneys.

Pathology.

The first change is hyperæmia of the intercellular and interlobular tissue, followed by the development of fibro-nucleated tissue from the alveolar and bronchial walls and the interlobular connective tissue. At the same time the epithelium of the alveoli undergoes proliferation.

As the new tissue contracts, it slowly replaces and obliterates the alveolar structure; and the new tissue development may reach such an extent that all the air cells of that portion of the lung, which is the seat of the process, may be replaced by connective tissue. The character of these changes, however, varies somewhat according to that of the inflammatory lesion from which they started. If the process originates as a peribronchitis, which is usually the case after ordinary catarrhal pneumonia or that induced by the inhalation of irritating particles, the proliferation and fibroid change around the bronchi gradually spread to the alveolar and interlobular septa. In most cases there is an overgrowth of vascular and corpuscular fibrous tissue, which spreads from the foci of irritation and involves the bronchi and the alveolar and interlobular septa; the alveolar walls thicken and any alveolar contents which are present eventually undergo organization; and finally, if the process continues long enough, extensive patches of consolidation are formed. When chronic pneumonia follows acute or lobar pneumonia the alveolar walls become primarily affected. These become thickened by small-celled infiltration, in which elongated, fusiform cells, resembling those found in embryonic tissue which is undergoing metamorphosis into fibrous tissue, may be distinguished. The growth contains blood-vessels in its earlier stages, but later, as the fibrous tissue undergoes contraction, these become obliterated. The growth, however, differs from the non-vascular one of phthisis in that it shows but little tendency to undergo molecular death and disintegration. The nuclei of the capillaries also undergo proliferative change. The alveolar cavities, after the fibrous tissue has contracted, either become entirely obliterated or else they are filled with exudation products.

The connective tissue is most abundant and most widely distributed when syphilis of the lung or other granulomatous inflammation is the etiological factor. In chronic pneumonia resulting from pleurisy, neoplasms, infarctions, etc., the lesion is more localized. In those cases which have their origin in the pleura, dense bands of fibrous tissue, which are developed in the interlobular tissue, pass inward from the pleura. No matter how the morbid growth may have arisen, it develops slowly, and in time involves the entire connective tissue of the lung. Like all morbid fibrous growths, it is cellular and well supplied with blood-vessels during its early stages, but sooner or later it becomes denser and tends to contract, so that in its more advanced state it often consists of closely packed wavy fibres or, more frequently, of a homogeneous or obscurely fibrillated structure containing a few round or fusiform cells.

The microscopical appearance varies with the extent of the fibroid

change. On section of the lung, there is a creaking sound with each movement of the knife; the cut portion is firm, hard, solid, and shiny; its color varies, it may be a dull red (red induration), bluish (slaty induration), and sometimes the lung has a marbled appearance. In that variety due to acute pneumonia there is generally only a slight thickening of the alveolar wall, the lung is more or less consolidated and presents the appearance of gray or red hepatization. When it is due to bronchopneumonia or pleurisy the process is localized in the earlier stages. In all advanced cases, however, the bronchi are more or less dilated. The dilated bronchi may form cavities of considerable size, and their mucous surfaces are often ulcerated or the seat of intense passive hyperæmia. Secondary inflammation and ulceration occur especially in the dilated portion, and appear to be due to the irritation of the retained and putrid secretion; the process may invade the indurated lung and so lead to more or less excavation. Gangrene may also be a subsequent process. The pleura on the affected side, except in the earliest stages, is always thickened. Induration of the lung, except in that variety which is due to the inhalation of dust particles, is generally unilateral; it may involve only a portion of the lung, when the apex is usually the site, or it may involve the entire organ. When due to the circulation of irritating particles it usually invades both lungs. The interstitial growth of fibrous tissue, caused by whatever the irritant may be in the bronchial tubes—chronic inflammation in the air vesicles or dust particles inhaled in the primary disease—having set up the changes in the blood itself, there is always accompanying it more or less abundant deposit of black pigment in irregular patches. This deposition of black pigment is not unusual in the lungs of persons of advanced age, but in cirrhosis of the lung it is often, if not nearly always, excessive in amount. The pigment is found in the thickened walls of the air cells, and especially in the connective tissue which surrounds the bronchial tubes and blood-vessels. The pigment separates the lobules one from the other. The lymphatic channels are also filled with the pigment, and it is always found in abundance in the bronchial glands. It is often found in the connective-tissue corpuscles. There is good reason to believe that it depends largely upon carbonaceous matter which has been inhaled into the lungs and has been absorbed by the mucous surface and then deposited in the tissues and taken up by the lymphatics. This pigment, which may at times be due to capillary hemorrhages and the resulting blood pigment, gives a peculiar mottled aspect to the surface of a cirrhosed lung when cut. At times the pigment may be present in such great quantity as to render the tissues uniformly and intensely black. The ultimate effects of this cirrhosis of the lung,

although it may perhaps, just as when occurring in the liver, produce a temporary enlargement, are finally a gradual contraction and diminution of the organ. The mucous membrane of the bronchi is itself congested and thickened. Bronchiectasis is usual from the contraction and retraction of the fibrous tissues surrounding the bronchia. The air cells, under the same circumstances, undergo gradual obliteration from the diminished cavities being frequently filled with disintegrating epithelial and other cells. Emphysematous enlargement of certain other cells may also be developed at the same time; for instance, should the cirrhotic process be limited to a comparatively small tract of lung tissue, an emphysematous process is by way of compensation established in the immediate vicinity.

Symptoms.

Inasmuch as this disease is a secondary one, its earlier symptoms are always disguised by the pre-existing affection; and it is not rare to meet with cases of extensive fibrosis in which the lung affection must have been present much longer than the symptoms accompanying it. If, after a lobar pneumonia or a bronchopneumonia has terminated, the dulness on percussion, bronchial breathing, slight elevation of temperature, cough, and dyspnœa continue beyond the period at which resolution should have taken place, the development of interstitial pneumonia may be suspected. This is usually the case in children, in whom it must always be suspected when resolution does not take place within the allotted time. It should be borne in mind that any long-standing inflammatory process of the lung may produce interstitial pneumonia, which must therefore always be looked for in these cases.

When the disease is fully established, its symptoms are more pronounced and are easily recognized. There are, however, cases in which the affection, by reason of its slow progress, the absence of bronchial catarrh, etc., may remain for a long time unrecognized. Usually, however, there are bronchial catarrh and bronchiectases, and these tend to make the indurating process more active. There are secondary inflammation and ulceration of the bronchial mucous membrane, retention and putrefaction of the secretions, and the consequent hectic fever. Dyspnœa is a constant symptom, relieved by lying on the affected side and aggravated by lying on the normal side. There is always cough, which varies with the quantity and character of the matter to be expectorated, the difficulty of its expulsion, and the susceptibility of the patient to the reflex influences upon which cough depends. If there are many bronchiectases, especially in the lower

part of the lung, the secretion accumulates within them, and its removal by expectoration becomes extremely difficult, hence the cough becomes violent and paroxysmal. A cough which occurs at longer or shorter intervals, is violent and paroxysmal, and is attended by a copious muco-purulent expectoration, is characteristic of bronchial dilatation in the lower portion of the lung. The expectoration is that of bronchial catarrh in the early stages. Later when there are bronchiectases and ulceration of the bronchial mucous membrane, great quantities of it are usually voided during the morning hours. It is then thin, muco-purulent or gray-black, and fetid. On standing it often separates into three layers: the lowest contains the solid matter, and is yellowish in color; the middle is greenish and fluid; and the surface is frothy, containing mucus and fat granules. If ulceration be present there is frequently hæmoptysis, but it is usually small in quantity.

Pyrexia, though usually present, is irregular and there may be long periods of complete intermission. Often the morning temperature is normal and the maximum evening temperature rarely exceeds 100°–102° F. The respiration and pulse are but slightly accelerated. As the disease progresses, there will be gradual loss of flesh and strength, night-sweats, dyspeptic symptoms, and frequently diarrhœa. The fever assumes the hectic type, and the consequent anæmia and interference with the pulmonary circulation may lead to cardiac hypertrophy and dropsy.

Physical Signs.

Inspection.—After the lung has contracted, there will be marked retraction of the chest wall. If the contraction invades large areas, the separation of the ribs, the eversion of the spinal column, the depression of the shoulder, and prominence of the scapula on the affected side are signs which make the diagnosis certain. The younger the patient the more the chest walls are retracted. There will be bulging and increased respiratory movement on the unaffected side. When the left lung is contracted, the displacement of the heart will be especially noticeable. The apex beat will be displaced upwards and outwards and its area will be markedly increased, due to the retraction of the lungs from the region over the heart and great vessels.

Palpation.—Vocal fremitus is usually increased over the affected side, though it may be diminished.

Percussion.—The signs furnished by percussion are those which represent solidification of the lung, the presence of air in dilated tubes or bronchiectatic cavities and emphysematous lobules, and they

are present in various degrees simultaneously in different portions of the lung or successively in the same portion. Solidification of the lung from induration, without dilatation or excavation, or if the cavities be filled with exudation, elicits dulness on percussion. The note is "toneless" or "wooden," high-pitched, and somewhat tubular, and a sense of resistance is imparted to the hand on percussing. The normal lung may overlap the diseased one; hence there may be a normal note anteriorly and a dull and wooden one posteriorly in some cases. Over a bronchiectatic dilatation a tympanitic resonance may have the amphoric or cracked-pot intonation.

Auscultation.—There is loss of the vesicular character in the respiration over the affected areas, and the breathing is more or less bronchial. With the respiratory sounds denoting consolidation, are associated bronchophony and the corresponding whispering signs, viz., whispering bronchophony and increased bronchial whisper. Râles are heard varying from the large, moist, metallic to the high-pitched bubbling ones. Sometimes they are only heard after coughing. Respiration on the unaffected side is exaggerated.

The symptoms may be briefly summarized as follows: In the case of a patient suffering with chronic pneumonia there is nearly always present considerable cough, worse during the winter months; upon the occurrence of catarrhal or pulmonary inflammation, the expectoration is generally not profuse, but if bronchiectasis is extensive it may be more abundant, and may even become fetid under those circumstances. During the summer months and warm weather the cough is very much better and the expectoration is much less. Should there be extensive bronchiectasis the expectoration may be excessively profuse, muco-purulent, or purulent in character, and if infected with the bacteria of putrefaction it will be exceedingly foul. If the cirrhosis is dependent upon coal-miner's lung or dust-lung, the sputum may be variously pigmented from the particles inhaled. Hæmoptysis is not of infrequent occurrence. In some cases the sputa may be more streaked with blood than in ordinary chronic bronchitis, while in other cases the hemorrhage may be quite profuse from time to time. This is due sometimes to the perforation of blood-vessels in the course of destructive changes which are taking place into the lung tissue, and at other times to the intense hyperæmia of the lining membrane of the dilated tubes. This hyperæmia is probably of an inflammatory nature or it may be from congestion owing to a weakened right heart. There is generally considerable pallor and lividity of the skin, as the disease progresses, with congestion of the tip of the nose and of the terminal phalanges of the fingers and toes. The pulse, which at first presents but little de-

parture from the normal, soon begins to decrease in volume and become rapid, weak, and irregular. When there is great obstruction to the pulmonary circulation, as is invariably the case in chronic pneumonia, the right heart dilates. At first there is considerable compensatory hypertrophy, but in the course of some years this gives way to dilatation, weakened circulation, pigmentation, œdema, and general anasarca. There is usually no very great elevation of temperature. As the disease progresses there may be developed at first hectic symptoms with occasional elevation of temperature, considerable perspiration from weakness, loss of appetite, occasional vomiting and diarrhœa, and gradual emaciation and debility. In short, the symptoms of cirrhosis of the lungs may very closely resemble those of chronic tuberculosis, except, of course, that the sputum does not contain the tubercle bacilli.

Diagnosis.

Interstitial pneumonia may be confounded with pleurisy with retraction, cancer of the lung, collapse, or pulmonary phthisis. In typical cases, where the affection is unilateral and has led to notable shrinkage of the entire lung, taking the physical signs in connection with the evidence of tolerance afforded by the symptoms, it is very easily differentiated. The differentiation from pleurisy with retraction is difficult. There will be retraction of the chest walls in both; however, in pleurisy the retraction is uneven and the ribs are twisted, whereas in interstitial pneumonia the retraction is more uniform, and there is a general diminution on the affected side. Fetid expectoration, hæmoptysis, and pyrexia may be present in chronic pneumonia but never in pleurisy. In interstitial pneumonia there is nearly always bronchial respiration, in pleurisy the respiratory sounds are feeble or may be entirely absent on the affected side. Though the physical signs of cancer resemble those of interstitial pneumonia very much, the history of the case is sufficient to differentiate the two diseases. Between phthisis and a typical case of interstitial pneumonia there is every degree of gradation as regards the combination of the anatomical characters of both. There is no great difficulty of differentiating them, however, except when the interstitial pneumonia involves only the upper lobes of the lung. Acute laryngitis and tubercular disease of the intestine argue against the presence of interstitial pneumonia; fetor of the sputa against that of phthisis. In unilateral basic disease and in disease of the whole of one lung, the question of phthisis can rarely present itself.

Prognosis.

Interstitial pneumonia is seldom a direct cause of death, and as a secondary disease the prognosis will depend upon the primary one. Extensive indurations of the lung and a chronic bronchitis may continue for years after bronchial dilatations have occurred. The hæmoptysis, marasmus, and right heart dilatation are accompanied by tricuspid regurgitation. It is hardly necessary to say that advanced interstitial pneumonia is incurable.

Treatment.

One of the principal measures in the treatment of chronic pneumonia should be the hygienic management of the case. A warm, equable temperature should be maintained in the patient's room if he is not able to go out in the sunshine. He should never be exposed to sudden changes of temperature. The air should be pure and wholesome and free from dust; the bodily strength should be maintained at its highest point of excellence, thereby preventing the supervention of complications. When the cirrhosis is due to his occupation, the patient should of course give up the pursuit. The cough and expectoration may require expectorants, the hæmoptysis, if at all alarming, will require astringents, and the other symptoms as they arise may require treatment. The patient should have daily outdoor exercise, but he should never exercise violently. His sleeping apartments should be warm and the atmosphere should be filled with the vapor of creosote at night. Prophylaxis is as important here as in other diseases. As chronic pneumonia is apt to occur after acute or bronchopneumonia, great care should be exercised in the later stages of those affections, with the object of gaining complete resolution. In the cases of lobar or lobular pneumonia which show a tendency to become chronic, marked benefit may often be obtained by residence in a warm, dry, and equable climate. In all cases the patient's general health should be made as good as possible. Great care should be exercised to prevent catarrhal troubles of the lung and therefore the patient must always be warmly clad. To improve the general health, cod-liver oil, iron, and arsenic are useful. The inhalation of turpentine, creosote, or carbolic acid often does much good in those cases which are associated with profuse expectoration. Expectorants of balsams with small doses of potassium iodide in syrup of poppy or syrup of codeine have proven beneficial.

Brown Induration of the Lungs.

Definition.—This is a chronic inflammation of the lungs produced by chronic congestion and consequent thickening of the walls of the air spaces, the cavities being filled with proliferated epithelial cells and a deposit of blood pigment.

Etiology.

Brown induration is caused by the mechanical obstruction to the blood returning from the lungs to the left cavities of the heart. Any valvular lesion of the heart with dilatation of the ventricles may produce this condition of the lungs; the most usual form is mitral regurgitation. Any mediastinal obstruction, such as that produced by tumors and growths interfering with the return of blood from the lungs to the heart, will produce similar results. The long persisting engorgement of the pulmonary circulation, from whatever cause, induces brown induration.

Pathology.

In mitral disease the left auricle is never completely emptied, and as a consequence the pulmonary veins cannot empty themselves completely. The result is that these vessels are constantly overdistended and this causes the capillaries in the air cells to dilate and become tortuous and their walls to become thickened. As a result the walls of the air spaces themselves gradually become thickened and pigmented. The congestion causes proliferation of the epithelial cells which soon fill the air spaces. As the air cells are granulating, so to speak, red blood cells escape and in the course of years of pulmonary congestion, proliferation of the cells, and exudation of red blood corpuscles, the portions of the lungs involved become chronically inflamed, developing a smooth, red hepatization due partly to extravasated red blood corpuscles and partly to epithelial cell granulation. The lungs become smaller in size from the contraction of the new fibrous formation. The pleural cavities may be dropsical and parts of the lungs may become œdematous. On section such lungs present a dense, tough, smooth hepatization or splenization. They are of salmon-pink color mottled here and there with brown or black spots. The larger pulmonary vessels, arteries and veins, show cloudiness and thickness of their internal coat as the result of the stasis. Beneath the pleura are found small dark spots of pigment with here and there a minute fresh hemorrhage. Microscopically examined the capillaries are shown to be dilated and twisted as a result of the persistent stasis.

The interstitial connective tissue is also found to be thickened and hypertrophied and filled with pigment granules which are the remains of extravasated and decomposed red blood corpuscles. This hypertrophic condition extends to the fibrous tissues of the parenchyma and may be seen down to the muscular tissue of the smaller bronchial tubes. After some time the internal coat of the smaller blood-vessels is found to undergo fibroid degeneration which may even extend to the muscular coat. A red induration of the lungs is described in which the infiltration does not seem to be so extensive as in brown induration. It would correspond to the stages of brown induration in which the lung is enlarged, red, and fleshy and beginning to be infiltrated with adventitious cell proliferation. Gray induration of the lungs is the name applied to the condition in which there is advanced cirrhosis. The lung tissues are extensively infiltrated with fibroid material; there is loss of blood supply, the organs are contracted, and as a consequence the tint is of a grayish hue. Black induration of the lungs is sometimes used to denote that form of cirrhosis in which the tissues have been largely infiltrated with black pigment, the result of capillary hemorrhages. The term also applies to coal-miner's lung in which carbonaceous particles have been inhaled and lodged around the alveoli and in the lymphatic spaces in the lung tissue.

Symptoms and Physical Signs.

The symptoms are generally referable to the cardiac disease. There is also considerable cough, with dyspnœa and muco-purulent or rusty sputum. There is often hæmoptysis. The breath sounds are interfered with, as the air capacity of the lungs is diminished. The lungs on auscultation abound in moist râles and there is considerable dulness, especially posteriorly if the patient continues for any length of time in the dorsal position. The hæmoptysis may be profuse, especially in that form of brown induration which depends solely upon mitral regurgitation. The hemorrhage is, however, seldom extensive, but the spitting of blood is sometimes constant, as the mucous membrane is intensely engorged, and capillary hemorrhages are apt to occur. There is also considerable dyspnœa, especially on exertion, and this gradually becomes more extensive as the disease progresses. It becomes quite serious on the occurrence of a fresh cold, additional catarrh, or any pulmonary inflammation. From the want of oxygenation of the blood the skin becomes pallid or livid, with congestion of the nose, fingers, and toes. The heart sooner or later becomes weakened as the compensatory hypertrophy gives way to

dilatation. The pulse becomes feeble, easily compressed, and the patient is apt to show signs of fainting on exertion. The cough is frequently quite distressing and the expectoration is often profuse, improving a little during warm weather and again becoming worse during the winter months. There is seldom much elevation of temperature unless some inflammatory action should supervene. The appetite is generally lost, consequently the patient becomes weaker and weaker. There may sometimes be albumin in the urine, which is scanty and highly colored. Œdema of the eyelids, feet, and ankles is frequently present owing to the interference with the circulation, and any unusual exertion is apt to prove too much for the heart on account of the obstruction to the circulation. As the heart becomes weaker and weaker pulmonary congestion and œdema occur and a fatal issue results.

Diagnosis.

Brown induration of the lungs is not difficult of diagnosis as the etiology is generally plain. The physical signs are also well marked. There is seldom any fever but considerable cough and expectoration; shortness of breath, dulness, and râles with the obvious valvular disease present. Whenever there are dyspnœa with more or less expectoration streaked with blood, considerable cyanosis, some anasarca, no fever but a weak and perhaps irregular pulse, and scanty, high-colored urine with some albumin, we may look for brown induration of the lungs, depending upon the changes in the heart, liver, or kidneys.

Treatment.

The cause should be removed, if possible, and the heart may be strengthened with strychnine, digitalis, and the ferruginous tonics. Stimulating expectorants may be useful, as may also diuretics and laxatives. Little can be done for the lungs themselves excepting to improve the condition of the circulation. In the case of acute congestion of the lungs much can be done in the way of relief. The condition of the heart can always be improved temporarily by means of strychnine, digitalis, strophanthus, etc. The lungs can be toned up by ether, atropine, alcoholic stimulants, and stimulating expectorants. The circulation can be equalized, in other words, a patient can be bled into his own veins (in order that the congestion and induration of the lungs may be relieved) by means of vasomotor dilators, such as nitroglycerin, aconite, veratrum viride, etc. A hot bath with a stimulating liniment applied to the chest will assist in relieving the engorgement of the pulmonary tissues. This plan of treatment should

be followed by diuretics such as potassium nitrate, digitalis, liquor ammoniæ acetatis, etc., and supplemented by saline laxatives, such as magnesium sulphate, sodium sulphate, and others. The action of the skin should be increased in serious cases, by the use of diaphoretics such as jaborandi and hot alcoholic drinks. In this manner acute congestions or indurations accompanying heart failure can frequently be relieved.

For the chronic condition nothing is better than the free exhibition of strychnine and ferruginous tonics with arsenic, easily digested food frequently administered, good hygienic surroundings, and plenty of fresh air and sunshine. Brown induration of the lungs, although a chronic disease, may, by the observance of the foregoing directions, be very materially benefited and life very much prolonged.

Pneumokoniosis.

Synonyms.—Anthracosis pulmonum, chalicosis pulmonum, silicosis pulmonum, siderosis pulmonum; miner's lung, miller's lung, baker's lung, etc.

Definition.—There are still many cases of pulmonary disease induced by the inhalation of dust particles, notwithstanding the numerous mechanical contrivances used to prevent the entrance of foreign substances from the atmosphere of the work-room into the lungs.

Etiology.

The lung diseases from dust inhalation comprise quite a long list. They are all principally confined to workmen whose occupation brings them daily into an atmosphere continually laden with particles of dust of different sorts. Workmen who daily inhale coal dust, as coal miners, charcoal burners, and graphite workers, soon contract anthracosis. Particles of carbon pass into the lungs in the inspired air and are deposited in the air cells and along the mucous lining of the tubes, and only a small portion is removed by the secretions and expectoration. The lymphatics absorb the remaining particles of carbon and carry them into the bronchial glands and tissues. In the case of persons living in large cities such as London or in manufacturing towns, the sputum is noticed to be dark or black, especially in the morning. During the winter of 1890–91 in London, when the fogs were quite severe, the writer constantly observed that the expectoration was laden with carbon particles, which could readily be seen under the microscope. On microscopic examination of the lungs of coal miners particles of carbon are seen in the lymphatic

channels surrounding the bronchioles and air vesicles. On post-mortem examinations in London of persons who have lived for a number of years in that city, the lungs are invariably found to be dark in color and under the microscope a large amount of carbon is seen deposited about the air cells and bronchioles.

Another lung disease is produced by the inhalation of the dust from flint, stone, marble, etc., as seen in those occupied in glass factories, stone and mill-stone cutters, stone and marble polishers, stone hammerers, plasterers, masons, slate quarrymen, potters, and workers in porcelain. This is termed *chalicosis or silicosis*, the *stone lungs*.

Still another occupation which produces serious lung trouble through the inhalation of dust is that of metal workers in such vocations as file-cutters, iron-workers, mirror-polishers, and grinders of knives and scissors. The dust consists of oxide of iron and fine particles of the metal itself. This is known as *siderosis* or *metal-dust lungs*. The writer has frequently observed *miner's lungs* in men employed under ground. Not only does the dust of the earth enter the lungs but also dust of the particular ore such as lead and sulphides. Frequently obstinate lead colic and salivation are produced besides the lung complications. Pulmonary disease is also produced by inhaling the dust in flour mills, bakeries, and candy factories, tobacco factories, saw mills, and cotton factories. One of the latest cases which has come under the writer's notice was in a healthy young man who was foreman in a jute mill in California. The dust from the jute had so completely broken down his health that it was necessary for him to retire from his position, and, although the patient has been under treatment more than six months, he is still suffering from chronic bronchitis of an obstinate character with beginning cirrhosis of the lungs.

Pathology.

The pathology of dust lungs depends somewhat upon the nature of the particles inhaled. Generally speaking, the dust slowly accumulates in the pulmonary tissues, bronchial and mediastinal lymph channels, and glands. At first it excites more or less irritation of the pulmonary mucous membranes, followed by considerable mucous secretion. The dust particles from smoke and coal are deposited in the interlobular septa beneath the pleural surface and around the smaller bronchi. Both lungs are generally equally affected. The apices may show the greater amount of deposit, or nodules may be scattered throughout the lungs. The same applies to chalicosis, siderosis, and other forms of dust lungs. The constant irritation sets up inflammation of the bronchial mucous membranes, this leads to interstitial in-

flammation and to the formation of connective-tissue overgrowth, resulting in diffuse chronic bronchitis, cirrhosis of the lungs, pulmonary emphysema, or bronchiectasis.

The chronic bronchitis prepares a favorable soil for the propagation of the tubercle bacilli whenever inhaled, and as a result tuberculosis frequently ensues. At times pneumonic spots and lymphatic glands break down forming abscesses. The dust lung on section is found to be studded with cirrhotic nodules, pneumonic spots, and abscesses. The tissues are of a bluish-black, reddish, or grayish color, depending upon the particular form of dust inhaled. Microscopically the tissues are found to contain the dust particles around the interlobular spaces, lymph channels, and glands. Chemical examination shows the presence of silicic acid, iron oxide, metallic iron, etc.

Symptoms and Physical Signs.

The symptoms are those of an irritative bronchitis with more or less profuse secretion. There is always some dyspnœa from the hypertrophied mucous membrane and considerable distress in breathing. The pain is usually not severe unless a pleuritis or pneumonia is set up. In chronic cases there may be considerable bronchial asthma, bronchorrhœa, bronchiectasis, emphysema, cirrhosis of the lungs, pneumonic consolidation, or phthisis, as a complication or sequel of the dust-produced disease. There is nearly always considerable hypertrophy of the heart as a consequence of the obstructed pulmonary circulation.

Diagnosis.

From the etiology, the occupation, and the symptoms of the disease superinduced by the pathological change in the tissues, the diagnosis is readily made. Otherwise the diagnosis is made from the existing condition of bronchitis, cirrhosis, or other changes induced by the irritating cause.

Prognosis.

This depends upon the severity of the pathological condition in the lungs. In the early stage of bronchitis the prospects are good if the vocation is changed, but when chronic changes have been instituted and pneumonia or phthisis supervenes the prognosis is bad, because there is no healthy lung tissue present. As a general rule such cases are not safe risks for life insurance, even if the disease is of short duration, as the normal physiological function of the lungs is difficult of reëstablishment, owing to the foreign particles in the

lungs, the choked lymph spaces, and the chronic interstitial fibrosis which is almost sure to develop. It is remarkable, however, how many persons become accustomed to the deleterious influences of dust inhalation. After the first bronchial irritation has subsided, the lungs seem to become more or less accustomed to the dust, and such persons will often continue work for many years without any serious consequences.

Treatment.

The cause should be removed by taking the patient away from his occupation. Tonics, good diet, plenty of sunshine and fresh air should be provided, and the pathological condition present must be treated then on general principles.

Pyæmia of the Lungs.

Pyæmia of the lungs is that condition of septic pulmonary inflammation which is connected with other unhealthy inflammation, such as sloughing bed-sores, necrosis or caries of the bones, puerperal fever, etc. It is characterized by infective emboli of the arterioles of the lungs and by the consequent occurrence of scattered patches of congestion, inflammation, hemorrhage, suppuration, or gangrene of the lungs. Sepsis occurs under two well-defined conditions. The first condition, known as sapræmia, depends upon the absorption of the chemical substances which are formed in the process of putrefaction and which soak into the surrounding tissues, acting like any irritant or alkaloidal poison. It is believed that pathogenic micro-organisms do not themselves produce sepsis, but develop septic ptomaines which are directly the cause of the disease. Sapræmia is the result of septicæmia, a disease depending upon the absorption of septic material into the blood without the formation of secondary abscesses in the internal organs. The poison given off by the micro-organisms is similar to certain vegetable alkaloids, such as strychnine, and produces toxic effects. Yet these micro-organisms themselves do not and cannot exist in living blood. This is typically exemplified in so-called puerperal fever, in which septic material is absorbed through the uterus, producing sapræmia, manifested by a severe chill, high temperature, 104° and 105° F., lasting for six to twelve hours, followed by profuse perspiration and great prostration; but in twenty-four hours the temperature is normal again. This condition depends upon the absorption of an alkaloidal ptomaine, similar in nature to certain of the vegetable alkaloids. Pyæmia, on the other hand, is an entirely different disease. Pyæmia depends upon infective micro-organisms which are themselves absorbed into

the blood where they live and thrive and multiply. These micro-organisms of pyæmia lodge in the various organs and tissues of the body, particularly in the lungs, and produce septic or infective emboli which cause inflammation, suppuration, and gangrene of the parts.

Etiology.

The etiological conditions out of which pyæmia of the lungs arises are necessarily numerous, as the lung tissue is merely one part of the anatomy liable to embolic infection. Pyæmia of the lungs is frequently set up in fractures of the long bones and bones of the head and pelvis. Surgical operations formerly were responsible for pyæmia of the lungs, but now one may operate in hospitals for many years without ever seeing a case. This of course is due to the modern practice of antisepsis and asepsis. In operations upon the prostate, urethra, or rectum, the parts being situated where it is almost impossible to render them aseptic, pyæmia or septicæmia may, however, occasionally occur. In operations on hemorrhoids, varicocele, or varicose veins, septic phlebitis may occur, giving rise to pyæmia of the lungs. Puerperal fever rarely nowadays causes pyæmia. Acute periostitis may set up pyæmia. Chronic diseases of the bones of the middle and internal ear are occasionally responsible for pyæmia of the lungs. Erysipelas and diffuse cellular inflammation, carbuncles, dissecting wounds, and the acute infectious diseases occasionally produce pyæmia of the lungs. The affection was formerly a very frequent disease in hospitals and in many lying-in institutions, but of late years it is practically unknown in such places. Chronic pyæmia, due to suppurating joints, portal pyæmia, ulceration of the intestine, hemorrhoidal lacerations, suppurating kidneys, gonorrhœal synovitis, or malignant endocarditis may occasion infectious embolism in the lungs. Ulceration of the mucous surfaces of the throat, cancer in various parts of the body, ulceration of the appendix vermiformis, malignant pustule, glanders, carbuncles, and boils are occasionally responsible for pyæmia of the lungs. Idiopathic pyæmia has been described, but the term "idiopathic," in the light of modern science, must be understood to mean the inability of the physician in charge to ascertain the cause of the pyæmia. This applies not only to pyæmia but to any and all other so-called idiopathic diseases. Pyæmia must of necessity have a definite cause, whether or not we are able to discover it. It is now generally recognized as being the result of suppuration due to the presence of the streptococcus pyogenes or of staphylococci.

Pathology.

Pyæmia most frequently spends its virulence on the lungs. Occasionally it finds its way to other organs. In persons dying from pyæmia we find scattered about near the surface of the lungs circumscribed patches ranging in size from that of a pea to that of a plum. These patches are sometimes distinctly hemorrhagic in nature and may be reddish-black or decolorized, partly broken down into a puriform mass. There are frequently patches of pneumonia surrounding these spots. These pyæmic spots often form abscesses or gangrenous cavities or they may remain as congested, inflamed, and suppurating cavities. Not infrequently general œdema and congestion of the lung tissue ensue or a diffuse pneumonia with consolidation may supervene. The pleura generally becomes inflamed near these spots, and subpleural petechiæ are common.

The pericardium, like the pleura, is often covered with extravasations of blood and pyæmic material; in fact, small yellowish patches of disintegrating tissue or abscesses may be discovered anywhere in the lungs or in any other organ of the body. Both endocarditis and pericarditis occur in the course of the disease. The liver, spleen, and kidneys are also occasionally infected, even the brain may become the seat of pyæmic emboli. The bones and the joints may become involved in a similar condition, and suppuration may manifest itself in the connective tissue and among the muscles of the body. When these infective emboli lodge in the blood-vessels of the lungs near the pleural surface, there is at first intense congestion, then inflammation, and soon there follow ulceration and suppuration of the embolic spot. The inflammation generally extends to the pleura, setting up an empyema. When an embolus lodges near the mucous membrane of a bronchus it rapidly leads to suppuration, ulceration, and submucous or gangrenous sloughs. When the embolus is laden with bacilli or micrococci, suppuration is soon established, and abscesses rapidly develop. Frequently when the veins bring these infective micro-organisms to the right heart, malignant endocarditis is set up. The left cavities of the heart may also be involved, although less frequently, so that we may have septic emboli of the pulmonary arteries and septic emboli of the bronchial arteries. On microscopic examination of these embolic abscesses of the lungs the micro-organisms of putrefaction are invariably found, thus establishing positively the cause of the pyæmia. The source from which comes the septic embolic material which plugs the pulmonary arterioles is either the right side of the heart or the systemic veins of the body. We

know that thrombi are formed in the veins in phlebitis of the lower extremities, in the pelvic veins, and in the endocardium of the right heart in the recesses between the trabeculæ, in the auricle, on the valves and the chordæ tendineæ, and at the apex of the ventricle. Small particles torn loose from thrombi so situated are carried along by the blood current until they reach the lungs and there plug larger or smaller branches of the pulmonary artery according to the size of the septic emboli. As the branches of the pulmonary artery are terminal arteries, the vascular territory belonging to each branch cannot be supplied with blood, excepting to a very limited extent, by collateral circulation from other vessels. The plugging of such a branch means closure of the blood supply, and the immediate result of the embolism is therefore an absence of blood from the territory supplied by the plugged vessel. This rapidly causes engorgement, as the vascular walls of the capillaries soon become abnormally pervious; and as there is a backward current into the region the fluid portion of the blood, the white blood corpuscles, and many red blood corpuscles rapidly penetrate through the walls of the vessels into the surrounding tissues and thus hemorrhagic infarctions are established. If emboli reach the lungs from an acute malignant endocarditis of the right side of the heart or from septic phlebitis, the pulmonary arterioles are plugged; if the left side of the heart is affected with rheumatic, syphilitic, or malignant ulcerative endocarditis, the bronchial arteries may be the seat of the embolic process.

Symptoms.

Pyæmia of the lungs is generally ushered in by a chill or distinct rigor followed by profuse perspiration. These chills come on irregularly, one or two a day, and are followed by high fever and profuse perspiration. The mucous membrane becomes congested, the skin assumes a sallow tint, the patient becomes dull, heavy, and restless, and behaves very much as one suffering from continued fever. The pulse becomes rapid, weak, and frequently intermittent. The tongue is glazed and at times fissured or coated in a similar manner to the tongue in typhoid fever. The lips are parched and the teeth become covered with sordes. The patient complains of thirst, loss of appetite, nausea, and vomiting, and frequently diarrhœa. The respirations become shallow and hurried. The inspiratory acts are attended with dilatation of the nares and separation of the lips. Cough frequently supervenes with considerable pain in the chest, and there are evidences of pleurisy or consolidation of the lungs with excessive secretion of the bronchial mucous membranes. As the

disease progresses there may be hæmoptysis and muco-purulent or purulent expectoration with distressing dyspnœa; the patient's face becomes shrunken and pale, and the mental functions are disturbed. Not infrequently there is considerable delirium, sometimes coma or convulsions, and death may ensue in from four to ten days in the acute variety of the disease. In chronic pyæmia of the lungs where an occasional embolus finds its way to the pulmonary tissues, the symptoms are less strongly pronounced, the fever assuming more the hectic character; isolated abscesses form here and there, but there is less expectoration and less hæmoptysis; there is less prostration—in fact the case resembles consumption. A peculiar and characteristic symptom of pyæmia of the lungs is the irregularity of the rigors. There may be one, two, or three a day and there may be none for one, two, or three days, depending entirely upon the formation of the embolic abscesses. The temperature is more or less characteristic after a severe chill and the deposition of one or more infective emboli in the lungs; it may rise to 104° and 105° or even 107° F., with corresponding restlessness and stupor. The respiratory acts, as the disease advances, become more frequent and show signs of pulmonary consolidation. Respirations often run as high as 50, 60, and 80 per minute. The breath acquires a peculiar sweet odor. There is always considerable bronchial expectoration, pain in the chest, pleuritic stitches, friction sounds, râles, rhonchi, and other auscultatory phenomena. The feebleness of the pyæmic pulse is more or less remarkable: at first it is generally rapid, full, and bounding, but it soon becomes feeble, irregular, and often reaches as high as 160 per minute. Besides the pleuritic friction sounds there may be pericardial friction and other signs of cardiac implication. The symptoms in pyæmia may be extremely grave, ending in profound nervous prostration and speedy death. In the more chronic cases there are anorexia, nausea or vomiting, diarrhœa, glazed tongue, sordes on the lips and teeth, marked weakness and prostration combined with restlessness, hectic, delirium, mental heaviness, and lethargy. The disease may last for some weeks or months. The patient gradually wastes away, the face becomes pallid and dull, there are hectic flush, a yellowish hue, shallow and hurried respiration, occasionally chilly sensations, and pyrexia. The temperature always remains above normal. We generally find albuminuria, capillary hemorrhages, hæmoptysis, muco-purulent expectoration, and gradual decay.

Diagnosis.

There is generally little difficulty in diagnosing pyæmia of the lungs when it arises after parturition or after surgical operations and

injuries. There may be more difficulty in establishing a diagnosis if the disease occurs during the course of some acute, chronic, or inflammatory infection as in erysipelas, carbuncle, diseases of the bones, diseases of the joints, typhoid fever, ulcerative endocarditis, etc. Some of the marked characteristics are the severe chills followed by high fever and great prostration. The evident involvement of the lungs is evidenced by the rapid breathing, dyspnœa, pain in the chest, hæmoptysis, muco-purulent expectoration, and great mental and nervous prostration, followed by delirium or coma. The disease comes on suddenly and develops rapidly. The patient may be perfectly well and in the course of a few hours, after a severe chill and high temperature, be taken very seriously ill. A careful search for etiological factors may also assist in establishing a diagnosis. Of course, if some unhealthy ulceration, bone disease, bladder infection, etc., exist, pyæmia would naturally be looked for. The chills recur at irregular intervals, the prostration comes on rapidly with mental apathy or delirium, vomiting and diarrhœa, dryness of tongue, feebleness and rapidity of pulse, and extreme muscular debility. All these symptoms come on much more rapidly in pyæmia than in any other disease. Differential diagnosis may be required between pyæmia and acute tuberculosis, acute ulcerative endocarditis, typhoid fever, acute rheumatism, pernicious anæmia, gall stones, Hodgkin's disease, and rapidly developing cancer. When pyæmia occurs in connection with obvious wounds, injuries, operations, or parturition there can be no question as to the diagnosis; but in many of the diseases mentioned it is necessary to carefully look into the etiological factors, course, duration, and symptoms of the existing conditions, with a careful examination of the lungs and in all cases a microscopic examination of the sputum. With these precautions a mistake in diagnosis is not liable to occur.

Prognosis.

The prognosis of pyæmia is more or less uncertain, but almost always exceedingly unfavorable. Recovery occasionally takes place; at the same time it is practically impossible in any given case to prognosticate recovery. Occasionally serious cases have passed on to resolution and recovery. The pyæmic spots in the lungs have undergone caseation and cicatrization and after some months or even years health has been reëstablished.

Treatment.

One of the first indications in connection with the treatment of pulmonary pyæmic, and for that matter of any other pyæmic disease,

is to remove if possible the source of infection. The next most important consideration is to treat the infective process antiseptically. The inhalation of oil of gaultheria, thymol, oil of cloves, turpentine, creosote, carbolic acid, or the like, is undoubtedly of benefit. For the high fever antipyretics must be used, but with discrimination. Cold may be applied to the chest by means of Leiter's tubes, the cold bath may be employed, and quinine may be given in ten-grain doses every few hours. Hot baths may be employed with the object of promoting perspiration; and purgation may be caused in order to aid the elimination of the poisonous material by way of the bowels. Sodium sulphocarbolate, in ten-grain doses every four hours, is of great service in pyæmic processes. Boric acid in from five to fifteen-grain doses has also proven efficacious in conjunction with five-minim doses of sulphuric ether every three hours. Salicylic acid in small doses is at times of great value in reducing the temperature during the first twenty-four or forty-eight hours of the disease, after which time it should not be used owing to its depressing influence on the heart. Permanganate of potassium in one-grain doses four times daily has been recommended. Antiseptic and stimulating expectorants may be employed with success.

Sulphites may be useful, and turpentine internally, in five-minim doses every hour or two for the first twenty-four hours, is highly extolled. Stimulants are necessary sooner or later. The best possible food should be given, with tonics such as mineral acids, quinine, tincture of chloride of iron, strychnine, etc. Occasionally it may be necessary to give large doses of opium to relieve the pain, but the drug should be used with care. After the first week or two it is important that no depleting measures be adopted, as the disease itself is very depressing.

Gangrene of the Lungs.

Definition.—Diffuse or circumscribed death and decomposition of portions of the lung tissue.

Etiology.

The cause of gangrene of the lungs is the entrance, under favorable conditions for their existence and multiplication, of the bacteria of putrefaction. Many people, if not most of us, daily encounter opportunities for inhaling these germs; but in health the human organism has the power of neutralizing and destroying them. In suitable cases, however, the micro-organisms rapidly invade the lungs, causing extensive death to the pulmonary parenchyma, and

putrefaction sets in. Gangrene of the lung is one of the terminations of acute pneumonitis. The engorgement becomes so intense that the inflamed tissues cannot receive sufficient nourishment and their death results. In acute miliary tuberculosis the bacteria of putrefaction find an excellent soil for their propagation. When there are cavities with profuse secretion the putrefactive elements have plenty of material to work upon. Gangrene may also supervene in consequence of cancer, abscesses, suppurating hydatids, from pressure of an aneurysm, foreign bodies in a bronchus, sepsis extending from the mouth, throat, or larynx, as in cancer, diphtheria, phagedenic pharyngitis, and sloughing tonsils. Septic particles may be conveyed to the lungs by the blood-vessels, forming embolic foci, as in ulcerative endocarditis, bed-sores, otitis, osteomyelitis, glanders, malignant fevers, and pyæmic processes generally. Particles of food or decomposing secretions in dilated bronchi may set up a necrosis of the lung tissue, especially in sufferers with bulbar paralysis and in the insane, who are apt to draw food into the bronchi and are unable to cough it up. Puerperal sepsis has produced the disease. Any acute inflammation, traumatic or septic, of the lung tissue may result in gangrene. The disease is much more prone to affect people of advanced years suffering with marasmus, intemperance, diabetes, and chronic wasting diseases. In dementia, epilepsy, and in severe nervous or mental cases gangrene of the lungs is not uncommon, and the exciting cause is more than likely the entrance of particles of food into the bronchi, with subsequent decomposition and septic infection. Bronchiectasis may also terminate in gangrene of the lungs. The stagnant muco-purulent secretion readily becomes infected and circumscribed gangrene results. Pulmonary gangrene may also arise from perforating cancer or ulcers of the stomach, œsophagus, and pleura, or from degenerating bronchial glands, and in rare cases of caries of the spine. The predisposition of patients suffering with diabetes to pulmonary gangrene is an etiological point which must always be borne in mind in these cases.

Pathology.

Circumscribed gangrene is the more usual form of the disease. It may involve a considerable part of a lobe, but the affected part generally varies in size from that of a hazelnut to that of a small orange. The lower lobes and superficial parts are most prone to become affected. The lung tissue affected soon becomes moist, soft, and pulpy, of a bluish or blackish-green color, and extremely offensive to the smell. From the circumscribed inflammation and septic in-

fection of the part, the blood supply is entirely cut off and death of the tissues is the result. There is generally a greenish-black core with degenerated, broken-down, stinking lung tissue surrounding. On pressure a foul-smelling fluid escapes. This product of decomposed lung tissue and septic material may escape through a bronchus during life, leaving a ragged sloughing cavity. At times the gangrenous spot discharges into the pleural cavity or, rarely, into the cutaneous cellular tissues when inflammatory adhesions have taken place. Embolic gangrene frequently locates itself near the pleural surface. From the infected centres new foci may radiate, setting up multiple gangrenous spots. Rarely the whole contents of the gangrenous area may be evacuated by a communicating bronchus and after a time, in favorable cases, the cavity will cicatrize and heal up. The blood-vessels which traverse these gangrenous areas seldom produce much hemorrhage, as the blood is already coagulated by the intense inflammatory process.

In the majority of cases of gangrene of the lungs the putrefactive micro-organisms enter the lungs through the bronchi by means of the air, particles of food, etc. Occasionally, however, the putrefactive material is transported into the lungs by the blood current. This is *embolic* gangrene and quite distinct in its causation and behavior. There are usually several spots or foci infected at or about the same time, and the process is more rapid and more serious than in the case of ordinary circumscribed pulmonary gangrene. Embolic gangrene of the lungs occurs in connection with extensive gangrenous bed-sores, puerperal processes, suppurative caries of the bones, and pyæmic processes generally. The putrid material enters a vein from the seat of the primary septic lesion and is conveyed to the right cavities of the heart and thence to the lungs. The venous putrid material often sets up malignant valvular disease, which is also a cause of embolic gangrene. Instead of an ordinary infarction the embolus, being infective in nature, rapidly sets up inflammation, suppuration, and gangrene. Embolic gangrene is generally found near the pleural surface of the lower lobes and more frequently on the right side.

The *diffuse* variety of pulmonary gangrene is not confined to any circumscribed spot but involves the whole of one lobe or one entire lung, setting up intense congestion, inflammation, and œdema; the blood supply is cut off and death *en masse* of the tissues takes place. The lung tissue rapidly becomes pulpy and softened and assumes the characteristic greenish or black color, with an abundance of dark-brown fetid fluid having an intensely putrid odor.

Symptoms and Physical Signs.

The general symptoms of gangrene of the lungs are unmistakable. There is extreme depression and adynamia with typhoid muttering, also marked evidences of nervous prostration, and signs of rapidly approaching collapse. At the onset there is usually a severe chill followed by high fever, inflammation of the lung, rusty sputum, and severe adynamia. The breath soon becomes offensive. There is usually considerable cough at first with the characteristic foul, frothy, partly liquid and partly muco-purulent, dirty-looking expectoration. Small brownish-black or greenish particles of gangrenous lung tissue will be discovered in the sputum, and the bacteria of putrefaction, leptothrix, fatty crystals, and elastic tissue will be found under the microscope. The cough is more or less paroxysmal and intense dyspnœa and prostration soon follow. There is usually some pain. The fever which was at first high soon becomes subnormal and there is evidence of systemic toxæmia. At times gangrenous particles in the sputum may be swallowed, setting up intestinal irritation and inflammation with severe tympanites and diarrhœa. Emboli are frequently carried to the brain, liver, spleen, kidneys, etc., which form infective foci, resulting in abscesses and gangrenous degeneration. Should the gangrenous area in the lung not communicate with a bronchus, the breath and expectoration may not be fetid, but systemic anæmia with dyspnœa and typhoid symptoms will be rapidly manifested and the gravity of the case will be made clear. Embolic gangrene, which is most frequently located near the pleural surface, often sets up a purulent and usually a sanious pleurisy by direct infection. When a gangrenous cavity opens through the pleura, a septic pneumothorax is the result. The expectoration in many respects resembles fetid or putrid bronchitis. When there is a gangrenous nodule the bronchi leading into it are generally infected, and a condition similar to putrid bronchitis is set up irrespective of the gangrene itself. When this putrid odor of septic bronchitis is present together with the repulsive odor of the gangrenous tissue the combination is about the most nauseating and disagreeable that can be imagined. The amount of sputum expectorated in twenty-four hours often reaches as much as ten or twenty ounces. When the sputum is collected in a glass, three distinct layers are formed on standing. The upper layer is muco-purulent and frothy, of greasy consistence and more or less watery in character; the middle layer is serous; the lower layer is almost wholly composed of pus and unctuous greenish-yellow plugs and shreds of lung tissue. These plugs are composed of needles of

the fatty acids, bacteria, detritus, and portions of the parenchymatous tissue of the lungs. The distinguishing test between fetid bronchitis and pulmonary gangrene lies in the finding of lung tissue under the microscope in the latter disease. On chemical examination of the sputum, the putrefactive organic matter is manifested by the presence of tyrosin, leucin, ammonia, sulphuretted hydrogen, butyric acid, caprylic acid, etc.

The *physical signs* will indicate the location of the gangrenous areas. Shortness of breath, cough and expectoration, dulness, bronchial breathing, and occasionally hæmoptysis, with the gangrenous odor from the lungs, will make the diagnosis clear. Many cases of gangrene of the lungs run an acute and fatal course without any hæmoptysis or bloody expectoration. Other cases, however, from erosion of the blood-vessels, severe inflammation, and ulceration may be characterized by profuse expectoration. There may be considerable concomitant pleurisy with effusion. To make a clear diagnosis of the character of the pleuritic effusion the harmless procedure of resorting to the use of the hypodermic needle to draw off a few drops of the fluid may be resorted to. In this manner the presence of a pneumothorax can also be positively established. Gastric and intestinal disturbances are occasionally witnessed in pulmonary gangrene, and must be due to the fact that part of the fetid sputum is swallowed after it has been coughed up. The putrid sputum affects the mucous membranes of the stomach, causing nausea and vomiting and in the intestines severe choleraic diarrhœa.

Diagnosis.

When there is fetid exhalation and putrid expectoration containing gangrenous lung tissue and elastic fibres, the diagnosis is easy. There are only two diseases for which this affection is liable to be mistaken, to wit, fetid bronchitis and bronchiectasis. But these latter diseases are exceedingly chronic in nature, and in them the sputum does not contain lung tissue but peculiar characteristic plugs. Gangrene of the lung is a more acute disease supervening in the course of pneumonitis, phthisis, cancer, abscesses, bed-sores, osteomyelitis, caries, suppuration, local or systemic pyæmic affections, in the aged or intemperate and in the insane, whereas bronchiectasis and fetid bronchitis are of slow progress and long duration without much fever or adynamia.

Prognosis.

Gangrene of the lungs is always exceedingly grave. Some cases of circumscribed single gangrenous spots may recover under appro-

priate treatment, but diffuse gangrene or multiple circumscribed cases are almost certain to prove fatal. The duration of the disease may be short, and its termination may be sudden in cases of extensive pneumonitis, embolic suppuration, and in the aged and insane, but frequently with proper treatment improvement or even recovery takes place. Death occurs either from general loss of strength from the gangrene itself or from the disease causing the gangrene, or from complications such as pulmonary hemorrhage, ichorous and purulent pleurisy, pneumothorax, multiple infective emboli in the brain, liver, kidneys, etc. Occasionally an infectious emphysema may rupture into the peritoneum, pericardium, or externally.

Treatment.

One of the first indications in gangrene of the lungs is to support the patient by administering abundant nourishment and large quantities of stimulants such as strychnine, carbonate of ammonia, Hoffman's anodyne, aromatic spirit of ammonia, tincture of iron, quinine, and alcohol. The next step is to encourage expectoration and to promote local antisepsis by means of antiseptic inhalations. For these purposes stimulating expectorants of ammonium iodide, squills, senega, serpentaria, or sanguinaria with the balsams may be used with benefit. The antiseptic inhalations which the writer has found most useful are turpentine, guaiacol, creosote, terebene, oil of cinnamon, oil of eucalyptol, compound tincture of benzoin, salicylic acid, boric acid, iodine, bromine, and pure oxygen. Internally, besides the tonics and stimulants, guaiacol, creosote, carbolic acid, sulpho-carbolate of zinc, bichloride of mercury, and chlorate of potassium have proven of value. Externally stimulating liniments with hot turpentine stupes and antiseptic poultices may be useful. The prime object must be to support and stimulate the patient. In cases of bulbar paralysis, and in general paralysis of the insane, great care should be taken to see that no food enters the lungs. In case of great difficulty in deglutition it is much better to feed such patients by means of the œsophageal tube. For internal use I have found the following valuable in pulmonary gangrene:

℞ Myrtol, ... ʒ ss.
Acetate of lead, ... gr. vi.
Creosote,
Oil of turpentine, ... āā ♏xij.
Extract of gentian, ... q.s.
Make into 12 capsules, of which one should be taken every third hour.

As there is frequently considerable gastric and intestinal sepsis

these myrtol capsules will answer the twofold purpose of being antiseptic for the digestive tract as well as for the pulmonary mucous membrane.

MALIGNANT GROWTHS IN THE LUNGS.

The malignant growths in the lungs include primary and secondary carcinomata, epitheliomata, sarcomata, osteosarcomata, lymphosarcomata, enchondromata, osteomata, and myeloid tumors. Primary malignant tumors of the lungs are rare; when they do occur they are nearly always carcinomata or epitheliomata growing from the columnar epithelium of the glandular crypts of the small bronchia.

Primary carcinoma usually grows slowly and forms a large dense whitish mass at the root of the lung. The extension is along the bronchial tubes, narrowing their calibre and at times extending into their lumen.

Secondary carcinoma occurs rather frequently from extension or metastatic deposition of cells from some other infected organ. The pulmonary growth then usually appears first on the pleural surface of the lung as thin grayish-yellow spots or patches, growing into nodules of various sizes and shapes. The primary growth is often in the breast, œsophagus, stomach, or liver. The lung may be largely converted into a mass of cancerous growth. The adjacent portions of the lung may become pneumonic or gangrenous. The cancer is generally of the soft or medullary variety. Sarcomata of the lungs are nearly always secondary, being deposited by the lymph spaces, or growing from direct extension of the primary disease. Sarcomata of the lungs often assume enormous proportions. They may grow from an osteosarcoma of the ribs, or be deposited from some distant organ in the abdominal cavity or from the testes. Lymphomata are generally also secondary, that is, other glands than the bronchial are usually involved. In pulmonary lymphoma, the glands following the tract of the bronchi or the interlobular septa become involved and infiltrate the lungs from the roots outwards.

Etiology and Pathology.

Cancer of the lungs is a rare disease. It occurs more frequently in men, notwithstanding that mammary cancer is found many times oftener in women. The cancer age is from forty to sixty years. Hereditary tendency to malignant disease no doubt plays an important part it its etiology. Cancer of the lungs is in the large majority of cases secondary to cancer of the bones and cancer of the testicles, but it may and does occur as a primary disease. The cylindrical-

celled carcinoma is the typical form found in the lungs. It grows from the bronchial epithelium and is exceedingly soft and vascular. Melanotic cancer is also occasionally observed.

Primary cancer is usually confined to one lung, especially the right. Secondary cancer usually involves both lungs. In secondary cancer the nodular form is most frequent, and both lungs are infiltrated, unless the disease occurs from direct extension. Cancerous nodules in the lungs are apt to undergo degeneration and softening from the surrounding inflammation and consequent shutting off of the blood supply. In this manner cavities are formed and considerable hæmoptysis and extravasation of blood into the tissues may occur. The vessels soon become involved, ulcerated, or obliterated by pressure. The pleura often becomes adherent and the tissues are all matted together. The general etiology and pathology of malignant tumors of the lungs are the same as those which govern cancerous disease generally.

Symptoms and Diagnosis.

The symptoms and diagnosis depend upon the location of the growths. Miliary disseminated pulmonary cancer cannot be diagnosed by physical signs. In cases of malignant disease of some other organ the presence of pulmonary complications should direct attention to the lungs. The glands in the neck and axilla should be examined. The sputum may also serve to establish a diagnosis. Generally in malignant growths one side is more affected than the other. The vocal fremitus is diminished. There is dulness over the cancerous mass, and the auscultatory signs show feebleness or absence of respiratory sounds with bronchial breathing over the site of the growth. There is marked dyspnœa, developing slowly, but steadily increasing in severity. There may be chronic inflammation of the lung about the site of the growth, with cough, muco-purulent or bloody expectoration, febrile movement, and pain, or a pleurisy with effusion supervenes according to the location of the growth. Cancer involving the bronchi usually produces obstruction. Primary cancer of the mucous membranes often causes death by profuse and repeated hæmoptysis.

When there is uniform infiltration of the lung the expectoration often resembles "currant jelly," although hæmoptysis is not very common. Pain is usually not severe. An important diagnostic point in cancer of the lungs is the enlargement of the bronchial, cervical, and axillary glands. These enlarged glands and the pressure from the growth itself may produce pressure symptoms, such as enlarged superficial veins, œdematous swelling of the head, neck,

and upper extremities. The vocal cords are also frequently involved, causing abductor paralysis from pressure on the recurrent laryngeal nerves. There may also be signs of dysphagia from pressure on the œsophagus. In any case the patient soon loses strength and flesh. He assumes the peculiar sallow cancerous cachexia, and as most of the cases are secondary the patient will exhibit the primary growth elsewhere. If a lung is extensively diseased there will be considerable enlargement of the affected side, with widened and flattened spaces. The surface may feel uneven although no fluctuation can be discovered. The heart and diaphragm may be displaced and there may be unusual cardiac sounds from pressure. When the lung is *infiltrated* there is usually contraction of the lung, with retraction of the chest walls and depression of the intercostal spaces. The non-cancerous parts of a lung generally show signs either of compensatory emphysema or of collapse.

Malignant diseases of the lungs either originate within the lung tissue or extend to it by continuity from the mediastinum or some other adjoining parts, or are secondary to a similar disease in some distant organ of the body. When the disease originates in the lung tissues it usually constitutes a solitary mass. When it invades the lung by continuity it usually advances along the bronchial tubes, and when the malignant disease extends to the lung by metastasis from some other organ the disease is for the most part multiple. When the disease extends along the bronchial tubes the connective tissue surrounding them becomes infiltrated and thickened and the tubes and blood-vessels penetrated, as it were, forming a solid mass of tissue. The bronchial tubes themselves soon become involved. The cartilages undergo malignant ulceration and the tube is soon eaten through, giving rise to severe hemoptysis and great prostration. Malignant diseases attacking the pleura or subpleural tissue generally appear in the form of small grayish thickenings something like drops of tallow on the skin. The disease generally separates along the interlobular septa and fibrous tissue, affecting isolated patches in the multiple variety or one lobe or one lung in the solitary variety. Malignant growths of the lungs, like those of all other organs, tend rapidly to undergo degenerative changes, hence they soon break down and form irregular cavities with considerable hæmoptysis. Not infrequently there are sloughing, expectoration of lung tissue, fetid breath, distinct cachexia, and marked depression. When the pleura is attacked first the symptoms resemble those of ordinary pleurisy. When the bronchial tubes are first involved the symptoms approximate those of bronchitis, or there may be symptoms of lung pressure with ulceration and hæmoptysis. There is generally con-

siderable expectoration of a muco-purulent character streaked with blood. The exact symptoms depend entirely on the location of the growth or growths. A cancerous mass may produce atelectasis, congestion of the lungs, œdema of the lungs, ulceration, or gangrene. Small malignant growths within the lungs seldom yield distinctive indications of their presence. When the tumor is large enough to be recognized there are of course dulness on percussion and the usual symptoms of lung pressure. There are always considerable dyspnœa and cough, and the expectoration may be mucous, muco-purulent, or hemorrhagic and fetid. There may be pleurisy, bronchial pneumonia, bronchial ulceration, abscesses or gangrene in the lungs, all depending upon the site of the tumor and the stage of its development.

As before mentioned, there may be pleuritic effusion. In such cases the aspirating needle will often furnish important diagnostic data. The frequent hæmoptysis, "currant-jelly" expectoration, implication of the glands, progressive emaciation, pressure symptoms, and cancerous cachexia, will usually enable the practitioner to make a correct diagnosis.

The *prognosis* is of course bad. Progressive emaciation and loss of strength generally reduce the patient to a skeleton, and death ends his suffering in from six to twelve months.

Treatment.

The treatment must be palliative, directed to relieving the pain, cough, and dyspnœa, and producing sleep. The toxins of erysipelas have been tried in these and similar cases, but as yet we are unable to speak encouragingly of the treatment.

SYPHILIS OF THE LUNGS.

That the lungs may be affected by tertiary and hereditary syphilis is abundantly proven by both clinical and pathological observations.

Pathology.

Syphilitic pneumonia, white hepatization, and so-called epithelioma of the lungs of new-born or very young infants are undoubtedly due to congenital syphilis. The disease presents a more or less diffuse infiltration of both lungs, which are distended, but the pleuræ are generally unaffected. The lungs affected with diffuse syphilis are heavy, dense, firm, and resistant, of a white or yellowish-white color. The affected parts become bloodless and the vessels disappear.

There is a considerable thickening of the alveolar walls and smaller bronchia. The walls of the vessels are also thickened with what seems to be a syphilitic fibrous overgrowth. One of the most characteristic features of the morbid anatomy of a syphilitic lung is the presence of gummata. These vary in size from that of a pea to that of a walnut, although they may occasionally attain the size of an orange. They are generally well-defined, rounded, encapsulated tumors of a grayish or brownish color, firm and dry in texture. In time they show the usual tendency to undergo caseation and formation of cavities. Microscopically they consist of fibre cells, abortive nuclei, and imperfect fibrous formation mixed with degenerative products.

Syphilis also produces a chronic interstitial pneumonia, leading to the usual fibroid infiltration and induration. The bases of the lungs are most frequently affected. In this form the disease seems to start from the pleural surface, dipping down into the lung tissue by means of fibrous bands. Such a lung presents a similar appearance to that of a fibroid kidney with superficial depressions and puckerings. At times the disease seems to start from the peribronchial structures, and leads to ulceration and gangrene. The mucous membrane of the bronchi also suffers from syphilitic infiltration and ulceration. When these ulcerations cicatrize they cause more or less obstruction or even complete stenosis of the tubes.

Syphilitic lesions of the lungs commence generally with proliferation of the connective tissue of the organ beneath the pleura, around the bronchial tubes and vessels, and in the interlobular septa, and terminate in ill-defined patches in which the tissues are indurated, grayish, contracted, and variegated, mainly in the fibroid or cicatricial tissue of the lung, studded in some instances with caseous or degenerated nodules. These caseous nodules in the lung as elsewhere, although having a close resemblance to tubercles, are usually much less friable and contain no tubercle bacilli or giant cells. The diseases with which syphilis of the lungs is most frequently confounded are chronic bronchitis, chronic phthisis, and cirrhosis of the lungs. Syphilis of the lungs may be confined, as in the new-born, to the pulmonary parenchyma, producing what is usually described as white hepatization of the lungs of still-born children, the origin of which is undoubtedly inherited syphilitic poison. These lungs or large portions of them are dense, yellowish-white, opaque, tough, and retain the impress of the finger when pressed. This syphilitic lung may also be seen in children who die some weeks or months after birth, presenting the characteristics of old people, the face filled with wrinkles, hair falling out, peevish, badly nourished, and generally unhealthy. The second form of syphilitic lung would seem to be an

acquired one—namely, the deposition of gummata in different parts of the lungs. The syphilis of the lungs which may be described as ulcerations of the mucous membrane of the bronchi is apt to lead to hæmoptysis and more or less bronchial constriction as these ulcers heal and the cicatrices contract. The pulmonary syphilis of the infant in which there is white hepatization is of less practical importance than the other forms of syphilis in which there are gummata forming in the lungs or ulcerations in the mucous membrane and bronchial tubes.

Symptoms and Diagnosis.

Syphilis of the lungs must be diagnosed by the history of constitutional taint and by the visible evidences of the disease. When the lungs become involved there is induration, usually of the lower lobes, circumscribed or general dulness, according to the form of infiltration or the existence of gummata. In the ulceration of the mucous membrane there is usually some hæmoptysis or obvious stenosis, dilated bronchi, cough, and bronchitis. The progress is very slow. Antisyphilitic treatment may assist in forming a correct diagnosis. There is generally neither fever nor pain.

Treatment.

The treatment is purely symptomatic outside of the usual antisyphilitic regimen. Large doses of the iodide of potassium or sodium with tonics will often clear up the lung and the diagnosis at the same time. For this purpose large doses of iodide of potassium or iodide of sodium with small doses of arsenic have proven very beneficial in the writer's hands. Twenty grains of the iodide of sodium with a thirtieth of a grain of arsenious acid are given one hour after meals in a suitable vehicle three times daily. At the same time the syrup of the iodide of iron, or, better still, the elixir of iron, quinine, and strychnine, may be given three times daily together with the best and most nutritious food that can be obtained. With specific treatment, ferruginous tonics, and constructive nourishment, much can be done toward curing acquired syphilis of the lungs.

BRONCHADENITIS.

Definition.—Inflammation of the bronchial lymphatic glands.

Etiology.

The lymphatics of the thorax frequently play an important part in the diseases of the lungs. The practitioner is apt to forget their existence unless his special attention is called to them now and again.

The deep lymphatic glands of the thorax are distributed as follows:— The intercostal glands are situated on each side of the spine near the costo-vertebral articulations; the internal mammary glands are placed at the anterior extremity of each intercostal space by the side of the internal mammary vessels; the anterior mediastinal glands are present in the loose areolar tissue of the anterior mediastinum, some lying upon the diaphragm in front of the pericardium and others around the great vessels at the base of the heart; the posterior mediastinal glands are situated in the areolar tissue in the posterior mediastinum, forming a continuous chain by the side of the aorta and œsophagus; they communicate on each side with the intercostal, below with the lumbar, and above with the deep cervical glands; the bronchial glands are situated around the bifurcation of the trachea and roots of the lungs. They vary in number from ten to twelve, the largest gland being placed opposite the bifurcation of the trachea, the smallest around the bronchi and their primary divisions for some little distance within the substances of the lungs. During infancy and childhood the thoracic and bronchial lymphatic glands present the same appearance as lymphatic glands in other parts of the body; in adult life they assume a greenish tinge, which in old age becomes a deep black color. The lymphatic vessels of the lungs consist of two sets, one superficial and the other deep; the superficial set is placed immediately beneath the pleura, forming the delicate minute plexus which covers the entire outer surface of the lung; the deep lymphatics accompany the blood-vessels and run along the bronchi. Both of these sets terminate at the root of the lungs in the bronchial glands.

These lymphatic glands, especially those of the bronchi, frequently undergo inflammation, enlargement, and even suppuration. The cause of bronchadenitis is usually scrofula or tuberculosis, in which affection the glands become enlarged and occasionally go on to suppuration. In syphilitic diseases the glands may also become enlarged, and in cancer of the lungs they are always involved; in lymphoma and in lymphadenoma the bronchial and thoracic glands are also frequently affected.

Pathology.

The pathology is essentially that of enlargement, inflammation, degeneration, and suppuration of lymphatic glands in other parts of the body. The disease is generally due to tuberculous infiltration, although occasionally, as just stated, the glands enlarge from syphilitic, cancerous, lymphoid, or sarcomatous growth.

Symptoms.

The symptoms of bronchadenitis are those of pressure and irritation, namely, cough, pain, spasmodic dyspnœa, dysphagia, and a peculiar change in the voice depending upon the pressure on the recurrent laryngeal nerve filaments. The general condition of the patient, taken in connection with the pulmonary complications, will assist us materially in arriving at important conclusions regarding the disease.

The usual obvious signs of cough and dyspnœa are absent in bronchadenitis as the disease is deeply seated. Generally, however, the superficial glands in the neck and other parts of the body will indicate the cause of the symptoms. The cough is dry, obstructive in character, and can often be excited by a deep inspiration. The pain is of an aching character and is referred to the root of the neck or between the shoulder blades. Spasmodic cough and dyspnœa often occur on exertion. The occurrence of dysphagia and alteration in the voice will depend upon the location of the enlarged glands.

Diagnosis.

The diagnosis must be made by exclusion and by the finding of other enlarged lymphatics. The spasmodic dyspnœa, cough, and pain, in the absence of any obvious cause, and the implication of the lymphatics generally will point to the diagnosis. The general cachectic condition of the patient in strumous or tuberculous cases, with considerable cough, pyrexia, etc., would indicate the trouble to be tubercular; the same applies to the cachexia of malignant diseases. In case of the suppuration and rupture of one or more of these glands into a bronchus or into the lung tissue there would be elevation of temperature, localized pain, and the expectoration of pus and glandular substance. Otherwise there is little if any expectoration and the symptoms leading to a diagnosis are those of pressure from obstruction. In severe cases there is collapse of the lung in some parts with compensatory emphysema in others.

Prognosis.

The course, duration, and termination of the disease depend upon the number of glands involved and the effect of treatment. In the majority of cases the cause of the affection is strumous or tubercular inflammation of the lymphatics and the prognosis is unfavorable.

Treatment.

The treatment must consist in the exhibition of alteratives and restoratives. Creosote in large doses (♏ x.) in wine or in an emulsion or by enema is productive of good results. Guaiacol in similar doses has proven beneficial. Arsenic and ferruginous tonics are indicated. Fresh pure air and wholesome food are indispensable adjuncts.

PULMONARY PARASITES.

Hydatids.

An hydatid tumor is derived from the development of embryos of the Tænia echinococcus.

Etiology and Pathology.

Primary hydatid tumors of the lungs are rare, but do sometimes occur, being found more frequently at the base of the lung than at the apex. Usually there is but a single tumor present, but there may be two or more. In size the hydatid varies from that of a pea to that of an orange, although the cyst may grow to occupy nearly the whole space of the thorax or abdominal cavity. The primary hydatid tumor of the lungs is situated within the substance of the lung or immediately beneath the visceral pleura. If in the latter position it generally forms an outgrowth into the pleural cavity. Hydatid tumors are usually solitary. The most frequent occurrence of hydatids in the lungs is by invasion from the liver and through the diaphragm. Hence the lower lobe of the right lung is most frequently affected. As the hydatid grows it displaces adjoining organs and interferes with their physiological functions. It finally ruptures spontaneously or in consequence of some unusual exertion, opening perhaps into a bronchus or into the pleural cavity. Sometimes it breaks into the pericardium or peritoneum, or externally. Occasionally a cyst is met with in which there are no echinococci; this is termed an *acephalocyst*. More rarely multilocular hydatid cysts are found.

Symptoms and Physical Signs.

The presence of hydatids of the lungs may be suspected if the liver or other organs of the body show signs of the disease. The expectoration or expulsion of hydatid membrane, fluid containing the characteristic hooklets, or echinococci themselves would afford the only positive proof. The lung tissue inflames, suppurates, and forms

cavities similar to those of phthisis. Occasionally an hydatid of the lung may undergo calcareous degeneration and its fluid contents be absorbed. This, however, is rare and only discovered *post mortem*. An hydatid cyst is always tense and of a globular or ovoid form, hence when it attains any considerable size it is certain on the one hand to cause circumscribed bulging of the thoracic parietes with more or less widening and protrusion of the extending intercostal spaces, and on the other hand to push aside the mediastinum and diaphragm. The circumscribed dulness on percussion will frequently pass beyond the median line of the thorax without extending to the summit. There may be no febrile or other acute symptoms, the general health of the patient remaining good. Such conditions are generally indicative of hydatid tumor of the lungs. Over this fluctuating tumor there will be an area of dulness and total absence of respiratory murmur and vocal fremitus. The only pathological conditions for which hydatid could be mistaken are circumscribed empyema or some solid tumor. Hydatid tumors may frequently undergo spontaneous cure, sometimes by retrogression, caseation, or absorption, and at other times by discharging their contents by way of the bronchial tubes or otherwise. On the other hand, a patient suffering from hydatid of the lungs may die suddenly from suffocation by the sudden discharge of the contents of the hydatid cyst; or he may prolong a miserable existence with long-continued suppuration of the lung or with an empyema due to the rupture of the cyst into the pleural cavity. From a diagnostic point of view it is frequently extremely difficult to distinguish between hydatid tumors of the base of the right lung and those developed in the upper part of the right lobe of the liver. The latter often protrude upwards far into the chest, displacing the diaphragm and base of the lung upwards, thereby causing marked bulging and dulness of the area usually occupied by the lower lobe of the right lung. By continuous pressure an hydatid cyst in the liver may perforate the diaphragm and open either into the pleural cavity or into the lung itself, causing empyema or profuse expectoration of the characteristic fluid and hooklets. When this occurs biliary coloring matter will usually sooner or later be expectorated with the contents of the hydatid cyst. An hydatid cyst of the lower lobe of the right lung may also penetrate the diaphragm and invade the liver.

Treatment.

The treatment must be conducted on general principles. It may at times be advisable to open the chest wall and wash out the cyst with peroxide of hydrogen. The treatment otherwise must consist

of restoratives, tonics, good food, and the best hygienic surroundings. If a tumor, whether an hydatid cyst or anything else, is so situated in the lung as to protrude and give evidence of its existence, there can be no possible harm in making an exploratory incision, as it may happen that the tumor may be entirely removed and health restored to the patient. Rather than to let a patient die from some supposed incurable disease it is better to make an attempt to cure by opening the thorax and thoroughly investigating the existing conditions.

Actinomycosis.

Definition.—The lungs are occasionally the seat of a fungous growth known as *actinomyces bovis* or ray fungus—a small, yellowish, glistening, spherical, granular mass about one-fortieth to one-tenth of an inch in diameter.

Bollinger (1877) was the discoverer of this fungus as the cause of disease in cattle. Israel, of Berlin (1877), and Ponfick (1878) were the first to call attention to the disease in man and to establish its identity with similar affections occurring in horned cattle and swine. Numerous cases have since been described in England and America. In man the disease frequently first invades the upper or lower jaw, at the site of a carious tooth. It may also gain entrance through the crypts of the tonsils and the pharynx. The disease spreads usually by direct continuity along the fascia. It may also spread by the lymphatics and wandering leucocytes (Roberts).

Pathology.

The pathology of actinomycosis in man is somewhat different from that observed in animals, although the micro-organism is the same. In animals the process is more in the nature of a local swelling, usually on the jaw. In man the disease tends to spread by direct extension and also by metastatic dissemination, and there is also a tendency to suppuration and the formation of multiple abscesses as is the case in chronic pyæmia. The explanation offered for this discrepancy in behavior is that the pure micro-organism finds its way to the animal, but when it reaches man it generally carries with it an admixture of pyogenic material which exerts a pyæmic influence. When the fungus enters the lungs it sets up a slow inflammatory process which was formerly considered scrofulous or tubercular in nature. This inflammatory spot soon begins to enlarge, forming slowly growing tumors in the posterior and lateral portions of the lungs, seldom if ever in the apices. The centre of the tumor is composed of the radiating fungus. Surrounding it is a thick layer of leucocytes and some of the club-shaped extremities of the radiating

threads of the fungus. The periphery of the tumor is composed of fibrous connective tissue. These tumors may grow to a diameter of two or three inches with several cavities or foci containing the fungus —threads and pus held together by a sort of cavernous tissue and trabeculæ of fibrous tissue. Surrounding these tumors the lung tissue is more or less congested and inflamed.

Symptoms and Physical Signs.

Actinomycosis of the lungs resembles chronic bronchitis, putrid bronchitis, pneumonia, or tuberculosis according to the stage of development of the disease. There are usually some fever, pain, and tenderness. The cough is apt to be paroxysmal and the expectoration muco-purulent, often foul-smelling and streaked with blood. The sputum after a few hours divides into two distinct layers (not three as in fetid bronchitis): the upper layer is clear and watery and the lower layer turbid, containing the typical granules and pus corpuscles. In the latter stages of the disease the expectoration becomes more copious, at times thick and viscid and at other times rusty and purulent. When the fungus lodges near the pleural surface of the lung, inflammatory lesions are set up causing pleurisy or pericarditis. Effusion often occurs, and both the visceral and parietal layers of the pleura not infrequently become involved, causing adhesion of the lung and extension of the disease into the muscles and skin. New foci establish themselves which break down and may discharge into the pleural cavity or externally. The discharge is muco-purulent or sanious, containing the characteristic granules. The fungus often finds its way from the lung into and through the diaphragm into the liver, the psoas and iliacus muscles, and the organs of the abdominal cavity. The disease is of slow progress, requiring months and years before a fatal issue supervenes. The cough and expectoration harass the patient. As in tuberculosis the patient suffers a general decline in health and strength, progressive emaciation, more or less cough and profuse expectoration, metastatic suppuration, night sweats, etc. Distant organs often become infected with the parasite, and abscesses may form in the liver, kidneys, intestines, heart, and brain.

Diagnosis.

The diagnosis of actinomycosis depends mainly upon finding the radiating club-shaped fungus in the expectoration or discharge. The migratory tendency of the affection is also characteristic, and we often find fistulæ which seldom heal but continue to discharge the typical granules. The disease is exceedingly slow in its course, now quiescent and then appearing with renewed vigor. The cough is very

troublesome owing to the difficulty of dislodging the micro-organism. In all suspicious cases of bronchitis the rule should be to examine the sputum, for by the microscope alone can we obtain certain evidence of many of the diseases which invade the lungs.

Treatment.

Medical treatment has hitherto availed but little. The fungus has great vitality, and once it gains a lodgment and begins to grow very few measures short of complete extirpation, promise satisfactory results. It is claimed, however, that a cure has been obtained by the free use of nitrate of silver internally. Caution must be exercised lest argyria be established. Favorable results have also been recorded by the free use of ferric sulphate, tincture of iodine, carbolic acid, and corrosive sublimate given internally and injected into the seat of the growth. Potassium iodide in large doses has also proven beneficial in some cases. Whenever an iodide is used, the acne eruption may be prevented by combining it with small doses of arsenic. It would also be wise to use ferruginous tonics as the iodide is very depleting. The latest alleged curative treatment of actinomycosis is by tuberculin, injected hypodermically as in the case of tuberculosis.

Distoma Westermanni.

A new parasite has recently been found in the lungs of cats, dogs, tigers, and man (Henry B. Ward, *Medical News*, March 2d, 1895). It is called *Distoma Westermanni* and is supposed to be of Asiatic origin. It reaches man probably through the inspired air, and is found chiefly in the pleuræ near the roots of the lungs. One patient is estimated to have expectorated no fewer than twenty thousand eggs every twenty-four hours for many years.

The distoma and eggs become encysted near the roots of the bronchi with which there is a communication, and produce anæmia, periodic hæmoptysis, and chronic cough with rusty and mucoid expectoration. These cases heretofore have been diagnosed as tuberculosis, but the microscope has revealed in the sputum large quantities of the yellowish-brown egg shells characteristic of the disease. The infected regions so far as known are Formosa, where it is said fifteen per cent. of the entire population are affected, also certain parts of China, Japan, and Corea. The disease has also reached America where several cases have been seen.

The *treatment* calls for removal from the infected district, the inhalation of creosote, menthol, or oil of cinnamon, with iron and strychnine tonics, generous diet, outdoor exercise, and free exposure to sunshine.

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